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A

PRACTICAL TREATISE

ON

URINARY AND RENAL DISEASES,

LANE LIBRARY

INCLUDING

URINARY DEPOSITS.

ILLUSTRATED BY NUMEROUS CASES AND ENGRAVINGS.

BY

WILLIAM ROBERTS, M.D., F.R.S.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON; PROFESSOR OF MEDICINE AT THE VICTORIA UNIVERSITY;
CONSULTING PHYSICIAN TO THE MANCHESTER ROYAL INFIRMARY.

ASSISTED BY

ROBERT MAGUIRE, M.D. Lond.,

MEMBER OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON; PHYSICIAN TO OUT-PATIENTS, ST. MARY'S HOSPITAL, LONDON;
LATE PATHOLOGIST TO THE MANCHESTER ROYAL INFIRMARY.

FOURTH EDITION.

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PREFACE.

The design of the present work is to give an account of the organic diseases of the kidney, and of those diseases and disorders of which the chief characteristic is some alteration of the urine.

The work naturally falls into three parts.

The first part, which may be regarded as introductory to the other two, is devoted to the physical and chemical properties of the urine, and to the various alterations which it undergoes under different circumstances of health and disease, in so far, and only in so far, as they seem to have a practical bearing. The methods of examining the urine for clinical purposes are explained; and the significance of the diverse changes experienced by it pointed out. The naked-eye and microscopical appearances of urinary deposits are described and figured, together with those of the extraneous matters which accidentally find their way into the urine.

Of the vast array of researches on the composition of the urine, and the rate of excretion of its several ingredients, accumulated in recent times, it has been found impracticable to give even an abstract without greatly exceeding the limits of practical utility. It has seemed to the author more convenient to consign these purely chemical and physiological materials to separate treatises, in the manner adopted by Neubauer and Vogel and Dr. Parkes, at least provisionally, that is, until such time as they can be shown to possess some clinical value. Further, these subjects are treated so amply in the works (in addition to those of the authors just mentioned) of Beale, Thudichum, Hassall, and others, that the omission of them has caused the author little regret. It is hoped, however, that nothing has been omitted a knowledge of which possesses any interest for the actual practice of medicine.

The second part treats of a group of affections which may be designated briefly as "urinary diseases," viz., diabetes insipidus, diabetes mellitus, gravel and calculus, and chylous urine. In his description of these diseases (with the exception of gravel and calculus), the author has endeavored to present an analysis of all the facts hitherto published in relation to them, together with those which have fallen under his own notice. In the chapter on gravel and calculus, prominence has been given to the medical treatment, and especially to the author's own researches in this direction.

The organic diseases of the kidney form the subject of the third and largest part of the work. The most important of these, Bright's disease and its allies, are treated with a fulness commensurate with their gravity and frequency, and mainly from a clinical point of view.

The less frequent affections of the kidney—hydronephrosis, cystic degeneration, cancer, tubercle, parasites, malpositions and malformations are treated analytically, and at considerable length. The extreme poverty of the older English systematic works on these subjects seemed to demand this compensation.

The third edition of this work has been exhausted for some years, but I have been hitherto unable, for want of leisure, to undertake the preparation of a fourth edition. Having, however, obtained the valuable assistance of Dr. Robert Maguire, this object has been accomplished. Dr. Maguire has carefully revised the entire work; and has brought up the several articles to the level of our existing knowledge.

I have almost entirely rewritten the articles on albuminuria and on microörganisms in the urine. Much new matter has been introduced into the chapters relating to Bright's disease. The observations of Bancroft, Manson, and Dr. Stephen Mackenzie on the filaria sanguinis hominis in relation to the causation of chyluria will be found incorporated in the chapter on entozoa in the kidneys.

The practical aim of the work has been steadily kept in view; and no alterations or additions have been introduced which are not designed to enhance the clinical value of the book. Nevertheless an endeavor has been made to give full references to pathological researches, with a view of facilitating the labors of special workers.

W. R.

MANCHESTER, 89 MOSLEY STREET,
January, 1885.

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INTRODUCTORY.

I.—SUMMARY OF THE PROPERTIES AND COMPOSITION OF THE URINE: ITS PHYSIOLOGICAL AND PATHOLOGICAL VARIATIONS.

HEALTHY urine is a clear, watery, amber-colored, saline solution, generally acid, with a specific gravity of about 1020. It contains a large quantity of *urea*; and smaller quantities of *uric acid*, *hippuric acid*, *creatine*, and *creatinine*. In addition to these, which are its characteristic constituents, the urine contains certain saline substances, namely, *chlorides*, *phosphates*, and *sulphates*, of which the bases are *soda*, *potash*, *lime*, and *magnesia*; also minute quantities of *oxalic* and *lactic* acids, *ammonia*, *pigment*, *diastase*,[1] and other substances which are classed under the head of *extractive matters*.

All these substances preëxist in the blood, and are simply separated therefrom by the secerning action of the kidneys.

The average proportions of the chief constituents of the urine

---

[1] It has been stated that traces of pepsin have been found in the urine. (Brücke, Sitzungsb. d. Wien. Akad., Bd. 43.) I have tested several specimens of urine with regard to this point, but have invariably obtained negative results. With regard to diastase the case is different. Healthy urines have a considerable power of changing starch mucilage into dextrine and sugar. This power is destroyed by heating the urine to boiling. I found the diastatic power of healthy urine to vary from 0.08 to 0.13, compared with healthy saliva as 10 to 17. That is to say, speaking roughly, urine has a diastatic value one hundred times less than that of saliva. (See the author's paper on "The Estimation of the Amylolytic and Proteolytic Activity of Pancreatic Extracts." Proc. Roy. Soc., 1881.) Grützner states that urine also contains rennet ferment and peptones, together with pepsin and trypsin, the last in the form of a zymogen. (Bresl. ärzt. Zeitschrift, No 17.

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All these substances preëxist in the blood, and are simply separated therefrom by the secerning action of the kidneys.

The average proportions of the chief constituents of the urine may be judged of by the following table, which has been constructed from a large number of the best analyses:

---

[1] It has been stated that traces of pepsin have been found in the urine. (Brücke, Sitzungsb. d. Wien. Akad., Bd. 43.) I have tested several specimens of urine with regard to this point, but have invariably obtained negative results. With regard to diastase the case is different. Healthy urines have a considerable power of changing starch mucilage into dextrine and sugar. This power is destroyed by heating the urine to boiling. I found the diastatic power of healthy urine to vary from 0.03 to 0.13, compared with healthy saliva as 10 to 17. That is to say, speaking roughly, urine has a diastatic value one hundred times less than that of saliva. (*See* the author's paper on "The Estimation of the Amylolytic and Proteolytic Activity of Pancreatic Extracts." Proc. Roy. Soc., 1881.) Grützner states that urine also contains rennet ferment and peptones, together with pepsin and trypsin, the last in the form of a zymogen. (Bresl. artz. Zeitschrift, No. 17.)

	Water	954.81
	Solid matters	45.19
	Urea	21.57
	Uric acid	0.36
Extractives	Creatine, creatinine Ammonia, hippuric acid Xanthine, hypoxanthine Sarcine, pigment, unoxidized sulphur and phosphorus, mucus, etc.	6.53
Fixed Salts.	Chlorine	4.57
	Sulphuric acid	1.31
	Phosphoric acid	2.09
	Potash	1.40
	Soda	7.19
	Lime	0.11
	Magnesia	0.12

The composition and physical properties of the urine may undergo alterations from *physiological* and from *pathological* causes.

PHYSIOLOGICAL ALTERATIONS.—The physical properties of the urine, and the relative proportion of its ingredients, vary greatly under the different conditions of healthy existence. Exercise, rest, the quantity and quality of the food and drink, digestion, fasting, sleep, the quantity of the cutaneous transpiration, atmospheric states, etc., react on the urine; and are, so to speak, reflected in its composition.

Some of the urinary constituents are derived, wholly or in part, directly from the food. This is especially the case with the saline or mineral matters, and the water. When the diet is especially rich, or especially poor, in any of these, their relative proportions in the urine rise or fall correspondingly.

Again, certain constituents (especially water) have other ways of passing out of the body than the kidneys, namely, by the skin, the lungs, the intestines; and if these show any unusual activity, the composition of the urine is necessarily affected. The greatest constancy of proportion is exhibited by the organic (nitrogenized) constituents—urea and uric acid, etc.—which are derived from the disintegration of the tissues; but even these oscillate not a little with the quantity and quality of the food, and with exercise or rest of the body. The reaction, which influences so importantly the physical properties of the urine, and its capacity for holding in solution certain ingredients which otherwise tend to be precipitated, is greatly affected by the digestion of food, and may be changed thereby from acid to alkaline during several hours in the day.

PATHOLOGICAL ALTERATIONS may be distinguished into *general* and *special*. It is desirable to indicate these separately; though practically they frequently merge into each other.

General pathological alterations are those which depend on some general bodily disorder, such as fever, rapid waste of the tissues, anæmia, etc. Alterations of this class, although of great interest for the elucidation of general pathological doctrines, have very little symptomatic value; and it has not been shown that a particular knowledge of them in an individual case of disease, is capable of furnishing any information on diagnosis, prognosis, or treatment, which may not be obtained more easily and accurately by other means, namely, by physical examination of the organs, temperature measurements, weighing the patient, etc.

Special pathological changes are: (*a*) those in which some new and unnatural ingredient is mixed with the urine—such as albumen, sugar, fat, cystine, blood, pus, fibrine, epithelial cells, spermatozoa, etc.; (*b*) those in which some constituent is present in such unnatural proportion that the circumstance forms a leading feature of some particular disease—as the excessive quantity of water in diabetes, the excessive diminution of urea in Bright's disease, etc.; (*c*) those in which some constituent is in an unnatural physical condition—thereby producing or indicating a particular morbid state—as in the occurrence of uric acid, oxalate of lime, and earthy phosphates as urinary deposits or calculous concretions.

In the present work, physiological and general pathological changes of the urine are only considered in so far as they possess some practical interest. The special pathological changes, on the other hand, are considered at length.

II.—METHODS OF EXAMINING THE URINE—APPARATUS REQUIRED.

An examination or analysis of the urine for clinical purposes is much more restricted in its objects than one which is designed for original investigations.

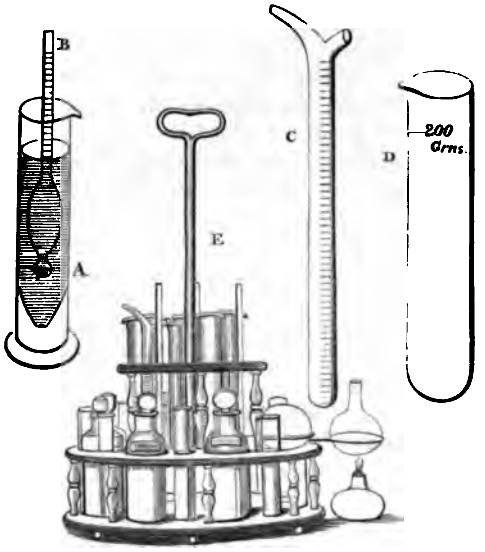
The object of the former is to ascertain those points, a knowledge of which, in a particular case, is found from previous experience to throw a light on the nature, course, diagnosis, prognosis, or treatment of the disease. The object of the latter is to obtain new and additional indications in the same directions; it embraces every conceivable information, and is consequently indefinitely elaborate.

The subjoined scheme is of the former kind, and is sufficiently simple to be within reach of every practitioner. It requires only an elementary knowledge of chemistry, and answers nearly all the requirements of actual practice.

The points requiring to be noted in an examination of the urine are—

1. The general appearance and color; clearness or turbidity; presence or absence of deposit, and of extraneous impurities.
2. Odor.
3. Reaction.
4. Specific gravity.
5. Presence or absence of albumen: if present, an approximate estimate of its quantity.
6. Presence or absence of sugar: if present, an estimate of its quantity.

FIG. 1.



Apparatus for urine-testing. A. Urine-glass—depth, 5½ inches; diameter, 1¼ inch. B. Urinometer. C. Burette. D. 200 grain measure. E. Stand of urine-tests.

7. An estimate of the total quantity of urine in twenty-four hours.

If there be a deposit, it is necessary to note—

8. Its aggregation and color: whether it be amorphous or crystalline, light or heavy; the manner of its subsidence or precipitation.
9. Its solubility or insolubility by heat; solubility in nitric acid, in acetic acid, in liquor potassæ; insolubility in both acids and alkalies.
10. By the microscope: absence or presence of crystals, their

appearance and form; of epithelial cells—renal or extrarenal; of blood disks; pus globules; spermatozoa: fibrinous cylinders; bacteria, etc.

The apparatus required consists of—

1. Three or four urine-glasses. Fig. 1, A.
2. Litmus paper.
3. Urinometer. B.
4. Half-a-dozen test-tubes.
5. Spirit-lamp.
6. Nitric acid.
7. Acetic acid.
8. Liquor potassæ.
9. Liq. ammon. fort.
10. Drop-tubes and stirring rods.

For sugar testing—

11. Prepared copper solution.
12. Graduated burette. C.
13. Two-hundred-grain measure. D.
14. Six-ounce graduated measure.
15. Small flask.

These may be conveniently arranged together for use on a circular stand of two tiers, as represented at E.[1]

A microscope is, of course, essentially necessary. It should be provided with a first-class ¼-inch object-glass, and an eye-piece to magnify not less than 240 diameters.

III.—EXTRANEOUS MATTERS IN URINE.

It is important that the student should be familiar with the appearance of certain extraneous matters which are apt to find their way into the urine after emission, and to be mistaken for urinary deposits.

Cotton fibres (*see* Fig. 2, *a*) have a flat limp appearance, are often folded on themselves, usually with a dark-looking medullary part; sometimes they present the appearance of narrow glassy cylinders. They vary in breadth from $\frac{1}{3000}$ to $\frac{1}{1000}$ of an inch. *Flax fibres* (*b*) are jointed at intervals, and have a round, solid appearance. Their broken ends are usually torn into a brush of fibrillæ. When sharply bent they break with a "green-stick" fracture. *Woollen hairs* (*c*) present the appearance of hard cylinders, with fine transverse markings and slight serrations along their margins. From their elongated form and somewhat similar diameters these three objects are liable to be mistaken for casts of the uriniferous tubes.

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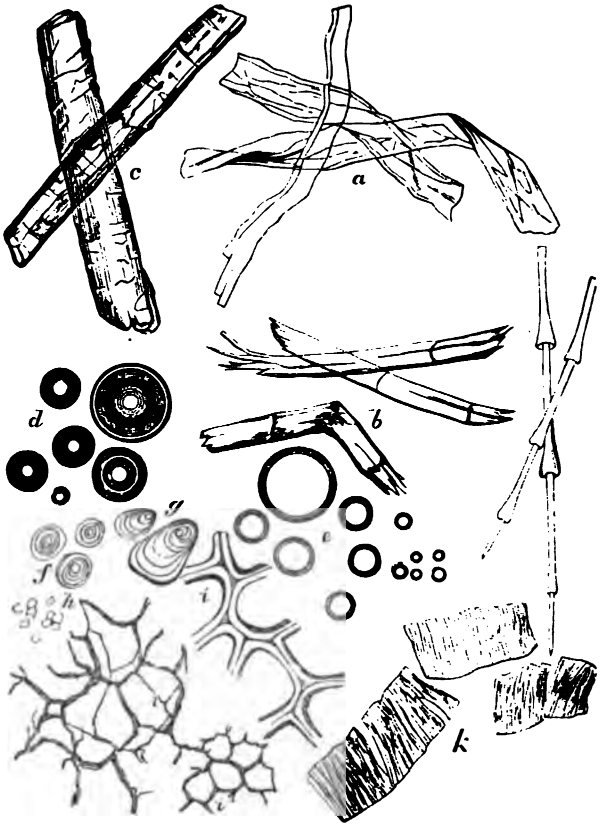
[1] This stand was constructed for me by Mr. Payne, of the firm of Mottershead & Co., Market Place, Manchester, from whom similar ones may be obtained, completely furnished, for the price of £2 2*s*. With the stand is supplied a printed card, containing directions for urine testing.

The latter, however, are distinguished by their softer aspect and less defined outline, and they are never fibrillated at their extremities.

A few air-bubbles (*d*) are generally retained beneath the covering-glass of the microscopic slide, and are apt to puzzle students.

FIG. 2.



Extraneous matters found in urine: *a*. Cotton fibres; *b*. Flax fibres; *c*. Hairs; *d*. Air-bubbles; *e*. Oil globules; *f*. Wheat starch; *g*. Potato starch; *h*. Rice-starch granules; *i i i*. Vegetable tissue; *k*. Muscular fibres; *l*. Feathers.

If small, they are spherical; if large, irregularly flattened. They are identified by their strong refraction, deeply colored thick borders and clear centres. Oil globules (*e*) are sometimes present in urine as a morbid product, in which case they are always very minute. More often they occur as accidental impurities;

they may be derived from the use of an oiled catheter; from milk, butter, broths, and other articles of food; from oily substances previously contained in the insufficiently cleansed bottle in which the urine has been conveyed for examination. Oil globules have a less strongly marked outline than air-bubbles; they appear flatter, and have generally a distinctly yellowish tint.

Confervoid vegetations or torulæ and various forms of bacteria are frequently encountered in the examination of the urine. The former are invariably derived from an extraneous source. The latter (bacteria) are sometimes of extraneous origin and sometimes are generated within the urinary passages. These will be more fully noticed hereafter. (*See* Microörganisms in the Urine.)

From the *sputa* may be introduced portions of bread, meat, fresh vegetables, as well as the epithelial débris of the oral cavity and air-passages. *Starch granules* find their way into the urine from certain articles of food, or the use of tooth and cosmetic powders. Wheat and potato starch granules are recognized by their concentric lines and hilus (*f g*). Rice granules are very minute cubical bodies (*h*). If the granules are ruptured by the operations of cookery (as in bread, puddings, gruel, etc.), they can no longer be identified by their forms, but a drop of iodine-water insinuated beneath the covering-glass instantly strikes a deep blue color with them. *Fecal matters* may mingle with the urine by inadvertence, or they may find their way into the bladder through a fistulous communication with the intestines. Their presence is recognized by the food remnants which they contain. At (*i*) and (*k*) are represented vegetable tissues and muscular fibres which were detected in the urine of a patient whom I saw with Mr. Jameson, of Heywood. The urine was not sensibly fecal to the smell; but the discovery of these structures in it proved decisively the existence of a narrow communication between the bowels and the urinary tract, and threw a strong light on an otherwise very obscure case. Particles of *soot* and *sand*, and other matters which may be designated as *dirt*, are of frequent occurrence. They are dark shapeless masses of various sizes, and all dissimilar. Any object of undefined shape, of which there are none similar to itself in the field, may almost with certainty be set down as dirt.

IV.—CHANGES IN THE URINE ON KEEPING.

The changes which take place in urine after emission are a frequent source of misapprehension. These changes differ in degree and direction according to the reaction and concentration of the urine.

A healthy acid urine generally undergoes the following series of changes. There occurs first a precipitation of the amorphous urates, then of uric acid and often of oxalate of lime. After a while confervoid vegetations or torulæ make their appearance. In the course of four or five days or longer the acidity begins to decline, and the urine passes into a state of ammoniacal putrefaction. It then becomes opaque from the development of myriads of bacteria; the odor and reaction of ammonia, together with an offensive effluvium of putrefaction, become perceptible. The amorphous urate deposit will now be found changed into dark round masses of urate of ammonia; uric acid crystals give place to bright prisms of triple phosphate and an abundant sediment of amorphous phosphate of lime sinks to the bottom of the vessel. The confervoid vegetations cease to grow with the change of reaction, and finally perish as the secretion becomes fairly putrid.

Urines of low density or of feeble acidity do not deposit urates on standing, and pass rapidly—in a day or two or even in a few hours—into a state of ammoniacal decomposition.

Under the name of the *acid urinary fermentation* Scherer[1] described a series of changes in healthy urine chiefly characterized by a progressive increase of its acidity, due to the production of lactic acid and partly of acetic acid. He attributed these changes to a fermentation in which the mucus of the bladder acted as a ferment on the urinary pigment, transforming it into lactic acid. Later researches by Röhmann[2] have thrown great doubt on the correctness of Scherer's account of the occurrence of a normal acid urinary fermentation. Röhmann found that in fourteen out of sixteen specimens of healthy urine no increase of the acidity took place. In the remaining two specimens a slight increase of acidity was observed; this he attributed to the presence of traces of sugar or of alcohol in the urine, both of them substances which readily yield acid (lactic or acetic) under the action of organized ferments.

The changes which take place in an opposite direction—that is, towards alkalescence—are much more prone to mislead than those just described. The transformation of urea into carbonate of ammonia (*see* Reaction) is a frequent source of confusion in the examination of the urine. This transformation is brought about with great rapidity by bacterial fermentation. The physical and chemical characters of the urine are then so altered, that it is unfit for clinical examination, and should invariably be rejected, except in cases where the transformation takes place within the urinary passages and a more natural specimen is therefore not procurable.

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[1] Annalen d. Chemie u. Pharm., Bd. 42, p. 171.

[2] Maly's Jahresbericht f. Thierchemie, 1881, p. 454.

In consequence of these changes it is desirable to examine the urine within a few hours of the time of emission. Certain organic deposits are liable to be greatly altered, or altogether destroyed, by an exposure of twelve or twenty-four hours, even when the more obvious characters of the secretion have not undergone a perceptible change. Blood corpuscles, renal epithelium, and renal casts, are very rapidly disintegrated, especially if the urine be of low specific gravity. On the other hand, pus, pavement epithelium, and spermatozoa resist much longer without effacement of their microscopical characters; and they may generally be recognized without difficulty in urine far advanced in putrefaction.

CHAPTER II.

PHYSICAL PROPERTIES OF THE URINE.

I.—ODOR.

THE natural odor of healthy urine is faint and peculiar; it may be described as *urinous;* it is due to the presence of certain volatile organic acids. The addition of a mineral acid greatly intensifies, and to a certain extent modifies, the urinous odor. The sense of smell is a rough test of the presence of ammonia, and of the freshness of the secretion, or the advent of putrefaction. When urine is alkaline from fixed alkali, it has a sweetish aromatic odor like that of the fresh urine of the horse or ox. In this way the smell of the urine comes to be a ready index of its reaction.

Certain drugs (turpentine, copaiba, cubebs), and certain articles of food (asparagus, garlic), communicate peculiar odors to the urine which lead to their immediate detection. Diabetic urine when fresh has a faint whey-like fragrance, and sometimes an odor resembling chloroform (*see* Diabetic Coma). When fermenting, diabetic urine smells like sour milk.

Urine containing blood or sanious discharges from the genital passages emits a stale, offensive smell, like the washings of slightly tainted flesh.

II.—COLOR.

The color of the urine in health is a yellowish-brown. It varies in intensity from the palest straw to a full amber. The study of urinary pigments is one of great inherent difficulty; and it has been rendered truly intricate by the multiplication of new terms by successive investigators, and the confounding of pigments produced by decomposition with those really preëxisting in the urine.

The coloring matters encountered in the urine may be divided into four categories, viz.:

1. Normal pigments of healthy urine.
2. Pathological pigments due to disease.
3. Derived pigments due to decomposition of the normal pigments, or of certain color-yielding extractives of the urine, especially in disease.
4. Adventitious pigments due to admixtures of bile, blood,

hæmatin, pus, etc., with the urine, or to the administration of certain drugs—logwood, rhubarb, senna, santonin, etc.

1. Normal Pigments of Healthy Urine.—Dr. Schunck's investigations[1] have led him to the conclusion that the ordinary color of normal urine is due to the presence of two substances having the properties of extractive matters. He has succeeded in separating these from one another, and from the other constituents of the urine. They have then the appearance of dark yellow syrups, being quite amorphous and deliquescent, with a peculiar, rather pleasant (not urinous) odor and a strong acid reaction, which proceeds from the presence of organic acids resulting from their spontaneous decomposition. The dilute watery solutions of these extractives have exactly the same color as urine itself.

The first of these extractives—which Dr. Schunck has named *Urian*—is soluble in alcohol and ether as well as water. Its composition is expressed by the formula $C_{86}H_{51}NO_{52}$, and does not vary. In a long series of experiments made with urine obtained at different times and from different places, Dr. Schunck always found its composition the same. It is decomposed at a boiling temperature, yielding a large quantity of a brown resin and volatile organic acids. Its watery solution becomes several degrees darker on the addition of sulphuric or hydrochloric acid, and a brown resinous substance is gradually deposited.

The second extractive he has named *Urianine*. Its formula is $C_{38}H_{27}NO_{28}$. This extractive is soluble in alcohol, but not in ether; it seems to have a great tendency to absorb four additional equivalents of oxygen ($C_{38}H_{27}NO_{32}$ = oxurianine), but without suffering any change in its physical properties. Urianine is probably a glucoside, for by the action of acids it yields a brown powder, insoluble in water (uromelanine), and the filtered liquid reduces the cupro-potassic test like grape sugar.

Urian and urianine are both decomposed when heated for some time in the water-bath, giving products which are insoluble in water. Watery solutions of both become several shades darker when mixed with dilute sulphuric or muriatic acid.

Jaffé has distinguished another pigment in the urine to which he has given the name of *Urobilin*.[2] Maly has shown that it can be formed from Bilirubin by reducing agents, and that it is identical with Hydrobilirubin. It is a red amorphous substance and soluble in alcohol, ether, and chloroform, partially soluble in water, having a characteristic spectrum, and giving in solution a green fluorescence.

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[1] Proceedings Roy. Soc., 1867.

[2] Virch. Arch., Bd. 47. See also a paper by McMunn in Proc. Roy. Soc., 1880, in which is given a process for the isolation of bilirubin.

Variations in the depth of the normal color of the urine correspond generally with its degree of dilution (or wateriness), and concentration. Very pale urines are voided by patients suffering from diabetes, from anæmia and chlorosis, and during convalescence from acute diseases—also by healthy persons after profuse drinking. Hysterical and nervous individuals, after paroxysmal attacks, void a very pale urine. As a rule, pale urines indicate the absence of pyrexia.

High-colored urines, on the other hand, accompany the febrile state, and any other morbid condition associated with rapid wasting of the tissues. Healthy persons void a similar urine after violent and prolonged muscular exercise and severe sweating.

The varying degrees of coloration of the urine have not as yet been made to yield much information of a practical value. Possibly this has been due to the want of an exact method of estimating and describing these variations. A first step towards this desirable object has been taken by J. Vogel, who has published a standard scale of colored plates, with which the color of any particular urine can be compared. Vogel divides the tints exhibited by urines into three groups, each consisting of three members. (*See* Plate I.) The first group consists of *yellow* urines, embracing: (1). pale yellow; (2) bright yellow; (3) yellow. The second group consists of *reddish* urines, and includes: (4) reddish-yellow; (5) yellowish-red; (6) red urines. The third group consists of *brown* or *dark* urines. These are subdivided into: (7) brownish-red; (8) reddish-brown; and (9) brownish-black.

In comparing the color of a urine with the tints of the scale, the two following precautions must be observed, in order to obtain uniform results. If the urine be not absolutely clear, it must be filtered. Secondly, the urine must be examined by transmitted light, in a glass which is four or five inches in diameter. Lastly, it must be remembered that the scale is not adapted for the estimation of the adventitious pigments sometimes found in urine, such as blood or bile; nor does it exactly reproduce some of the pathological tints observed in disease.[1]

2. Pathological Pigments.—The most familiar of these is a reddish-pink pigment (purpurine of Bird, and uro-erythrine of Heller), which makes its appearance in various febrile and other complaints. Purpurine has an intense affinity for uric acid and

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[1] The use of Vogel's scale for the purpose of estimating the *quantity* of pigment in the urine is, I believe, impracticable; and the statement made by him that all the nine varieties of color form one continuous series, and that they "may be considered as merely different degrees of dilution of one and the same pigment matter," is certainly, according to my experiments, inexact. The real usefulness of the scale consists in the aid it gives to accurate description.

the urates, and when the latter are thrown down as a deposit it communicates to them a beautiful pink color. Purpurine abounds in the urine of persons suffering from severe organic diseases, and especially organic diseases of the liver; it is likewise present in all febrile and inflammatory urines. It is said to be abundant in poisoning by lead and other metals.

A black pigment, melanin, is excreted in the urine of patients suffering from melanotic tumors, especially when the disease attacks the liver or the skin. The fact was pointed out as early as 1820 by Norris, and afterwards, in 1826, by Fawdington, a Manchester surgeon. Occasionally the urine is pale when passed, but becomes darker and deposits a black or brown precipitate on exposure for some time to the air, or immediately on the addition of a strong acid. Thus in the fresh urine there is present a colorless substance, melanogen, which on oxidation yields the dark pigment. Zeller[1] has lately reported a very interesting case of melanotic tumors of various organs, in which the quantity of melanin in the urine varied in inverse proportion to that of urobilin. He argues, therefore, that the two coloring matters must belong to the same group of bodies.

In certain other conditions the urine shows the peculiarity of being pale when passed, but becoming dark in color on exposure to the air. (*See* p. 48.) Pyrocatechin, which is always present in the urine of the horse, has been found by Ebstein and Müller and other observers in human urine in rare cases. The urine may then show the above reaction, but the dark color appears immediately if caustic alkali is added. Bödecker described, under the name of alkapton, a body in the urine, which other researches seem to show to be identical with Pyrocatechin.

The so-called alkapton urine reduces Fehling's solution, almost like diabetic urine, but differs from it in not fermenting with yeast. In a case described by Dr. Armstrong (*Dublin Journal of Med. Sci.*, 1882), Dr. Smith found that the urine, while presenting the above characters, contained not pyrocatechin but protocatechuic acid.

[A specimen of urine sent to me lately by Dr. Gray, of Armagh, also contained protocatechuic acid. The patient was a young lady, under the care of Dr. Gray and Dr. Whitla, of Belfast, and had suffered from symptoms of ulcer of the stomach, with apparently perforation and consequent attacks of localized peritonitis. At intervals she passed urine which, while of ordinary color at the time of leaving the bladder, became of a deep brown, almost black color, after standing for a short time. The urine as I received it, that is, after being corked up for some time, was of a pale brown color and of an alkaline reaction, and contained a deposit of triple and stellar phosphates. There was a somewhat more than normal amount of indican present. If the urine were exposed to the air for a short time, or if a little liq. potassæ were added, a deep dark brown color was produced. When, however, the urine was acidified, it might be exposed for some days without any alteration in color occurring, and the dark urine became considerably lighter on the addition of acetic acid. The urine showed only a slight reducing action on Fehling's solution, but immediately reduced an ammoniacal solution of nitrate of silver. A weak solution of ferric chloride, when added to the urine, caused the appearance of a bluish-green coloration, which, on the addition of a little liquor ammoniæ, changed to reddish-violet. These reactions showed the presence of either pyrocatechin or protocatechuic acid. The peculiar coloring agent was, however, not distilled over on heating, nor removed by shaking with ether, and hence, according to Dr. Smith, it must consist of protocatechuic acid and not pyrocatechin. The patient passed the dark urine only at intervals, but it was found that between these periods the urine was acid, and occasionally, at least, if alkalized, it showed again the change in color. It is evident from the above reactions that this dark coloring matter was a product of oxidation, but that it could only be formed in an alkaline solution.—R. M.]

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[1] Arch. f. klin. Chirurg., Bd. XXIX. p. 245.

3. Derived Pigments.—Schunck has shown that the normal pigments of the urine are extremely susceptible of decomposition. All strong alkaline or acid reagents, and even simple boiling, are sufficient to change them; and there is little doubt that a considerable number of the substances described by previous writers as pigments preëxisting in the urine, were, either partly or wholly, products of such decompositions. Among these may be enumerated the various *brown* and *blackish resins* of authors, the *melanic acid* of Prout, the *uræmatin* of Harley, and probably the *urochrome* of Thudichum.

The discovery of *indican* (or a substance closely resembling it) as a normal constituent of the urine by Schunck, afterwards confirmed by Carter, has thrown a strong light on the nature of some of the pigments found in the urine. This substance, which appears to be identical with the uroxanthine of Heller, imparts a yellow color to the urine, and yields, by decomposition, two colors well known in the arts, viz., *indigo-blue* and *indigo-red* (uroglaucine and urrhodin of Heller). Indigo-blue is frequently seen in putrescent urines, forming glistening blue shreds and films on the sides of the glass and the surface of the urine. Occasionally it is observed clinically. It was so noticed by Prout, who clearly indicated its nature and composition. It is also probably identical with the cyanourine of Braconnot. In the highly ammoniacal urine of cystitis I have seen on two occasions the precipitated urate of ammonia tinted of a beautiful violet by indigo-blue. The quantity of indican in urine varies from a mere trace to a considerable proportion. For its detection an equal part of hydrochloric acid is mixed with the urine, and afterwards a concentrated solution of chloride of lime is added drop by drop, and the mixture well shaken. The indican is then decomposed, forming indigo-blue, which imparts a greenish or blue color to the mixture (Jaffé). According to Senator, if a little chloroform or ether be now shaken up with the solution, this will take up the indigo, and on standing will form a separate layer. The depth of color in this layer will then give an approximate estimate of the amount of indican present. Normal urine treated in the above manner is usually colored reddish-violet. The quantity of indican in the urine is increased in various affections, the more important of which are obstructions in the intestines, peritonitis, and cancer of the stomach. The subject has been studied, experimentally and clinically, by Jaffé, who has found that obstruction in the small intestine and diffuse peritonitis are accompanied by great increase in the amount of indican, while obstructions in the large intestine and circumscribed peritonitis cause only a slight increase. Senator has observed increase of indican in the urine in many chronic affections, accompanied by changes of nutrition. He has also found a similar increase in cases of granular kidney, but not in other forms of Bright's Disease.[1]

4. ADVENTITIOUS PIGMENTS.—In jaundice the coloring matter of the *bile* is freely excreted by the kidneys, and communicates to the urine a color varying from a saffron-yellow to a dark olive-green. Bile-pigment in urine may be discovered by placing a few drops of the secretion on a white porcelain plate, with a few drops of nitric acid in juxtaposition. The two fluids are brought into contact by inclining the plate: if bile be present, a beautiful play of colors—violet, green, and red—is observed, which passes rapidly away. Bile-pigment appears in the urine before the skin is perceptibly discolored; it also continues after the skin has attained its natural tint; so that its recognition is sometimes a useful warning of impending jaundice or a verification of a preëxisting jaundice. When a urine containing bile is kept for some days, it sometimes changes to a grass-green color from oxidation of the biliary pigment.[2]

Dr. Harley considers that the presence of the biliary acids in the urine is characteristic of jaundice from retention of bile, as distinguished from jaundice arising from suppression of bile.

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[1] Salkowsky has described a great increase of carbolic acid in the urine in cases of obstruction in the small intestine. Normal urine contains only a trace.

[2] Maréchal has recommended tincture of iodine as a test for bile-pigment in the urine. It gives a green color, passing to rose and yellow.

For the detection of the biliary acids he recommends that a couple of drachms of the urine be poured into a test-tube with a small fragment of loaf sugar. Then about half a drachm of strong sulphuric acid should be slowly added, in such a manner that the two fluids shall not mix. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing a few minutes—*a deep purple hue.*[1] In a case of long-standing retention of bile from compression of the common duct by a cancerous growth of the head of the pancreas, which I saw with Dr. Henry Simpson, only a brandy-red coloration of the urine was produced by the application of this test.

Blood and *pus* mixed with the urine communicate to it their appropriate colors. (*See* Hæmaturia, Hæmoglobinuria, and Pus in Urine.)

Certain *medicinal* and *poisonous* substances administered internally produce peculiar alterations of color in the urine. Creasote, and the external application of tar ointment, have been known to produce a very *dark*, almost *black* urine. In some cases of this kind which occurred in Guy's Hospital, Dr. Odling identified the dark coloring matter with indigo-blue, and he pointed out the close chemical relations between indigo and creasote.[2] The application of carbolic acid solutions to the surface of wounds also causes the urine to become very dark, and occasionally, although the urine is light when passed, it becomes darker on exposure to the air. Baumann and Preusse[3] have shown that this is due to the presence in the urine of hydrochinon, a further oxidation product of carbolic acid. It is excreted as a sulphate which is colorless, and very easily decomposed. The free hydrochinon then absorbs oxygen from the air, and forms a dark pigment which gives the peculiar color to the urine. Marcet, Prout, and Dulk have also described cases in which a black coloring matter existed in the urine. In patients taking gallic acid a dusky hue is communicated to the urine. Vogel records an instance of black discoloration of the urine after poisoning by arseniuretted hydrogen. (*See* Hæmoglobinuria.)

Rhubarb given internally colors the urine a deep gamboge-yellow, which is changed to red by the addition of ammonia. *Senna* communicates a brownish, and *logwood* a reddish tinge to the urine when administered as infusions. *Santonin* imparts a conspicuous orange-red color to the urine if it be alkaline, and a rich golden-yellow if it be acid.

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[1] Harley on Jaundice, p. 61.

[2] Bird's Urinary Deposits, 5th ed., p. 336.

[3] Zeitschrift f. Phys. Chemie, III. p. 156.

III.—DENSITY OR SPECIFIC GRAVITY.

The specific gravity of the urine is estimated by means of the urinometer. The instrument indicates whether the urine is concentrated or dilute: and as the range of health is very great, the density does not yield direct indications of disease; nevertheless the information thus furnished is in some cases of great importance, and indicates at once the path of further research.

The usual range of density in healthy urine extends from 1015 to 1025; but it frequently mounts above or sinks below these limits. After abundant potation on an empty stomach the urine is profuse in quantity, clear, and dilute as water. Under such circumstances the density may fall as low as 1000.6, and numbers varying from 1002 to 1008 are common. Copious drinking on a full stomach has comparatively little immediate effect on the flow of urine. Prolonged fasting renders the urine concentrated. How high it is possible for the density to mount in healthy individuals it is difficult to say; but I have known it as high as 1036. With this very considerable range in health, caution must be exercised in drawing inferences from any unusual depression or elevation of the density in disease. If, however, the urine exhibit habitually, and especially in the morning before breakfast when the urine is naturally concentrated, a density below 1015, the presence of albumen in it may be suspected; if the density persist at a still lower point—1005 to 1008—the existence of insipid diabetes is to be apprehended. After hysterical paroxysms in women, after similar attacks in men, and sometimes in the apoplectic state, the urine is discharged in large quantity and of exceedingly low density. In cases of suppression of urine from mechanical obstruction, the urine—if any escape past the obstacle—is of remarkably low density. This peculiarity will be treated of more fully in a future chapter. (*See* Suppression of Urine.)

On the other hand, a density above 1025, especially in a pale, *apparently* dilute urine, is strongly suspicious of the presence of sugar; and the higher densities, from 1035 to 1050, belong almost entirely to saccharine diabetes. Yet, not exclusively so; the heaviest urine ever submitted to my examination, which had a density of 1065, did not contain a particle of sugar, but a very large quantity of albumen.

A high density in a urine free from sugar indicates concentration, and more particularly a large percentage of urea. In the febrile state there is an absolute increase of urea, uric acid, and the sulphates in the urine, with a diminished elimination of water, consequently the specific gravity ranges high. The urine has also a high density when there is rapid wasting of the tissues, especially if there be concurrent sweating or diarrhœa, in simple abstinençe after profuse perspiration from any cause, and after excessive ingestion of nitrogenized food without a corresponding use of aqueous fluids.

From the density of the urine, a rough estimate may be formed of the percentage of solid constituents contained in it; and if the quantity voided in twenty-four hours be known, the daily excretion of what may be called "solid urine" can be approximately ascertained. Tables have been constructed on an experimental basis, exhibiting the quantity of solid matters per 1000 parts in urines of different specific gravities; and formulæ have been proposed by means of which the same result can be obtained by a simple calculation. Probably the most accurate, as well as the simplest formula, is that proposed by Trapp. According to this, if the last two figures of the specific gravity are doubled, the quotient represents the amount of solid matters per 1000. A thousand grains of urine, sp. gr. 1020, would therefore contain 40 grains of solids.

This method yields but rough approximations. If the urine were a solution of a single substance, or of a number of different substances in a fixed proportion to each other, the rising and falling density would indicate accurately the varying strength of the solution; but the urine is a fluid of complex composition, and its numerous constituents vary every hour in their mutual proportions, so that the results obtained in this way cannot be regarded as exact estimates. Vogel took the trouble to inquire what are the precise limits of error in this method; and he assigns them as follows: In healthy urines there is a liability to r or of $\frac{1}{10}$ or even $\frac{1}{7}$; but in morbid urines, and especially those of high density, the range of error may reach $\frac{1}{5}$ or even $\frac{1}{4}$. With very multiplied observations this method certainly yields results of practical value; and it is the only one which can be used by practitioners generally. When more accurate results are required, resort must be had to the tedious but more exact method of evaporation to dryness, and weighing the residue.

From a large number of observations by different physiologists, Dr. Parkes estimates the mean discharge of solid urine in healthy men, between twenty and forty years of age, living on good diet, at 945 grains per day.

IV.—QUANTITY OF THE URINE.

Closely connected with the specific gravity, and holding an inverse relation to it, is the quantity of the urine. The mean daily discharge ranges, in health, between 40 and 50 fluid-ounces. There are, however, considerable differences between individuals. The average for some persons is only 35 ounces a day; for others as much as 67 ounces. Oscillations in the same individual on different days are also very considerable. The urine may mount to 70 or 80 ounces, or sink to 25 ounces within the limits of health.

The flow of urine is essentially regulated by the quantity of fluid drunk: controlled, however, in a most important degree by the pulmonary and cutaneous exhalation, and by the call of the system for water at the time. When the blood and tissues contain their full complement of water, any further potation results in immediate diuresis, whereby the superabundance is carried off. But when the organs and tissues of the body are craving for more water, a large quantity may be drunk without causing diuresis. The kidneys eliminate water in strict accordance with these conditions—it being an essential and important part of their function to regulate the aqueousness of the blood.[1]

There is very great irregularity in the flow of urine from hour to hour as the conditions of its separation vary. After prolonged fasting the urine may sink to 2½ drachms per hour; during sleep, likewise, the urine flows slowly—at the rate of about half an ounce per hour; but after meals it rises to two or three ounces; and after drinking abundantly on an empty stomach I have seen 26½ ounces secreted in an hour; so that the stream of urine may run 85 times stronger at one time than another. It would seem, indeed, as if the kidneys (in health) supplied conditions of an almost mechanical nature, by which they were enabled to separate water at an almost unlimited rate—equal, at least, to the capacity of the gastric vessels to absorb water.

When the mode of life is equable, and the meals are taken at regular intervals, the quantity of urine secreted at different periods of the day and night follows certain tolerably regular oscillations, as is shown in the following table, which is a fair sample of a very large number of observations:

Breakfast at 8; dinner at 2. Sleep from 11 P.M. *to* 7 A.M.

Time of day.	7–8 A.M.	8–9	9–10	10–11	11–12	12–2 P.M.
	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.
Hourly rate.	0 6	1 0	2 0	1 4	1 7	1 3

Time of day.	2–3 P.M.	3–4	4–5	5–6	6–7	7–9	9–11	11–7 A.M.
	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.
Hourly rate.	1 2	10 0	2 3	2 3	2 9	1 4	1 0	0 4

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[1] The experiments on which these and the remarks which follow are based, are fully detailed in two papers by the author—one in the memoirs of the Manch. Lit. and Phil. Soc., 1858–9; and the other in the Edinb. Med. Journ., March and April, 1860.

A much closer insight into the varying activity of the kidney is obtained by comparing the quantity of *solid urine* excreted at different periods of the day. The solid matters are much more constant in their quantity than the volume of the urine, which is liable to be greatly affected by potation, perspiration, etc. The annexed table contains the average results of observations made during seven days, all consecutive except one. The solid urine was calculated from the specific gravity in the manner explained in the preceding section:

Time of day.	Solid urine discharged per hour, in grains.	Diet, etc.
8– 9 A.M.	29.27	Breakfast at eight; coffee or tea, with meat and bread and butter.
9–10 "	39.22	
10–11 "	44.34	
11–12 "	45.24	
12– 2 P.M.	41.43	
2– 3 P.M.	38.69	Dinner at two; meat, potatoes, bread, cheese, water.
3– 4 "	38.79	
4– 5 "	41.21	
5– 6 "	41.09	(No solid food of any sort taken after dinner.)
6– 7 "	49.01	
7– 9 "	47.44	
9–11 "	37.66	
11– 1 A.M.	28.53	
1– 7 A.M.	15.53	Hours of sleep.
7– 8 A.M.	17.75	Prolonged fasting in the waking state.

The table shows in an interesting manner the increase of the renal excretion after meals, and its diminution during fasting and sleep. The increase began within the first hour after breakfast, and continued during the succeeding two or three hours; then a diminution set in, and continued until an hour or two after dinner. The effect of dinner did not appear until two or three hours after the meal; and it reached its maximum about the fourth hour. From this period the excretion steadily decreased until bed-time. During sleep it sank still lower, and reached its minimum—being not more than one-third of the quantity excreted during the hours of digestion.

All the urinary ingredients appeared to partake in the increase after meals. The urea was found more than doubled; the uric acid more than trebled; the earthy and alkaline phosphates nearly doubled.

The table shows that the vegetative functions share to some extent with the animal in the repose of sleep. The mean hourly discharge of solid urine during the waking hours, on the seven days of the table, was 33.14 grains; while the average of the hours of sleep was 15.53 grains, or less than one-half. This difference is not, of course, to be wholly attributed to the effect of sleep, inasmuch as, under the arrangement of meals, during this series of observations, the period of sleep was also a time of fasting. A more exact estimate of the effect of sleep alone is obtained by comparing the urine secreted during the hours of sleep with that secreted during hours of combined waking and fasting. If we take the last two hours before sleeping (from 11 to 1), and the first hour after waking (from 7 to 8), we shall find that the mean discharge of solid urine in these three hours was 23.59 grains per hour, which is one-third more than the average of the sleeping hours.

In drawing practical conclusions concerning any deviation from the usual volume and quantity of the urine, the following points should be borne in mind.

When the urine is unusually *scanty*, it should be ascertained, before pronouncing it a morbid phenomenon, whether the patient has abstained from liquids above his habit, whether water has been eliminated in excess by some other channel, as the skin or bowels. The urine is always scanty in cirrhosis of the liver; in some forms of Bright's disease through their entire course; and in the last stage of all forms; in any condition of the heart which directly or indirectly causes passive congestion of the renal veins whereby the circulation through the kidneys is impeded. In the early stage of acute Bright's Disease, the urine is very scanty, sometimes approaching or reaching total suppression. The same occurs in the collapse period of cholera. Partial or total suppression also occurs in the later stages of all organic diseases of the kidneys; and when any mechanical obstacle obstructs the flow of urine. A diminution of the urinary secretion which at all approaches suppression is of most serious consequence, and is soon followed by a formidable train of symptoms, which bring life to a termination unless speedily relieved. (*See* Uræmia.)

The flow of urine is *abundant* when the surface of the body is cool; also as a direct and invariable consequence of potation, unless some of the conditions already mentioned intervene.

In disease, the urine is discharged in excessive quantity in two special maladies—diabetes insipidus and diabetes mellitus, which will be described in future sections; also in the middle stages of atrophic degeneration of the kidneys. Temporary excess of urine occurs after hysterical paroxysms, and certain other convulsive attacks in males and females. An increased tension in the arterial system, as in some cases of hypertrophy of the left ventricle, is associated with increased secretion of urine. It is a curious circumstance, that in several organic diseases of the kidneys in which the renal substance is gradually destroyed (atrophic Bright's Disease, cystic degeneration, double hydronephrosis), the volume of the urine is sometimes increased though the solid matters are diminished. This appears to be an attempt on the part of Nature of a compensating character, to maintain, by excessive transudation of water, the depurating function of the kidneys under failing anatomical conditions. When at length the destruction has gone so far that this kind of compensation can no longer suffice, symptoms of fatal suppression of urine rapidly supervene.

V.—SUPPRESSION OF URINE (ANURIA).

Suspension of the secretion of urine arises under two distinct classes of circumstances. (1) It may arise from organic disease of the secreting tissue or from some disturbance in the innervation or vascular supply of the kidneys. These cases are not dependent on any impediment to the outflow of the urine by its excretory channels, and I would propose to classify them under the designation of *non-obstructive suppression.* (2) The secretion of urine may be suspended by the establishment of a mechanical obstruction in the ureter or in the pelvis of the kidney. In these cases the organs themselves are not primarily at fault, but their function is arrested by the blocking up of their excretory channels. These may be designated as cases of *obstructive suppression.*

The two classes of cases contrast with each other, not only in their determining cause, but also, most markedly, in their symptoms and course. Non-obstructive suppression rarely, if ever, proves fatal as a direct consequence of the suspension of the secretion of urine, but through the general effect of the shock and collapse which have produced that suspension. The cases usually run a very rapid course, ending in death or in recovery in a few hours or a day or two. Suppression from obstruction produces its effects more slowly; if it result fatally, the termination is delayed for eight or ten days or more. In both classes of cases the suppression is frequently not absolute; some small quantity of urine is generally voided, and the character of this urine is distinctive. In non-obstructive cases the urine is either high-colored and concentrated, or it contains albumen and casts, which plainly indicate disease of the renal tissue. In obstructive cases, on the other hand, the urine which escapes past the obstacle is pale and watery, and devoid of albumen and casts.

1.—*Non-obstructive Suppression.*

There is comparatively little known about this subject, and it merits a more extended study.

In the terminal stage of chronic Bright's Disease, the urine is often suppressed for ten or twenty hours before death, probably from the destructive process in the kidney having reached an extreme degree.

In the earliest stages also of acute Bright's Disease, especially in the scarlatinal form, the urine is sometimes suppressed for many hours or for a day or two. The return of the urinary flow is sometimes sudden and very copious, more frequently it is gradual. A similar suppression may occur in scarlatinal dropsy, which is unaccompanied by albuminuria. The following is a characteristic example:

A child of seven had sickened with scarlet fever about a fortnight before my visit. The attack was mild; and the patient entered on convalescence five days ago. Two days ago general anasarca set in, with vomiting and purging. The urine became scanty, almost to suppression. When I saw the child only two drachms of urine had been voided in the previous twenty-four hours. It was of a deep saffron color, and highly concentrated; it contained casts, but not a trace of albumen. The pupils were strongly contracted, and the tongue and skin were dry. There were vomiting and purging. Under the influence of blanket baths, a considerable amelioration in the general symptoms was produced; but in the two following days only three ounces of a deep yellow urine were voided. On the fourth day violent vomiting came on again, and loud pericardial friction was heard. Death took place next day—preceded by great restlessness, but without coma or convulsions. The total quantity of urine voided in the last seven days of life amounted to between six and seven ounces. No autopsy was permitted.

The most remarkable examples of this form of suppression occur in that state of system which is called shock or collapse. In the algide stage of cholera and yellow fever the urine is frequently suppressed for twenty or thirty-six hours. The return of the flow is generally gradual; the first portions of urine being scanty, high-colored, and containing blood and albumen.

All types of violent fever and inflammation are liable to be complicated with suppression of urine. And a similar effect may attend an overdose of turpentine or poisoning by the mineral acids and other irritants. Riegel has described diminution in the quantity of urine during paroxysms of lead colic.[1]

Suppression also is a common attendant on the shock and collapse following serious internal injuries, such as rupture of the bowels or of the liver, spleen, or uterus.

The kidneys appear peculiarly sensitive to injuries or violence applied to the urethra, and rapidly fatal collapse with suppression of urine has been known to follow slight operations on the urethra, or even the passing of a catheter. I remember an instance in which death in twenty hours—with total suppression—followed catheterism in an old case of stricture where instruments had been repeatedly used before without any ill-effects. The only post-mortem appearance of consequence was intense congestion of the kidneys. Sir H. Thompson mentions a case in which a man with old-standing narrow stricture died fifty-four hours after the passing of an instrument which had been used at least a hundred times before. No damage whatsoever was found to have been inflicted on the urethra. Rigors and vomiting commenced about an hour after catheterization, and not another ounce of urine was secreted from that time until death. In this case the kidneys were found congested to an extraordinary degree, but no sign of inflammation existed in any part of the excretory urinary apparatus. Similar consequences have been known to follow Holt's operation for stricture. In Mr. Fayrer's case rigors and vomiting ushered in collapse, with total suppression of urine, and death in thirty-six hours.[1]

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[1] Deutsches Arch. f. klin. Med., Bd. 21, pp. 175, 193.

Renewed attention has recently been called to this group of cases by Sir A. Clark in a paper read before the Medical Society of London, entitled "Catheter Fever." (*See Brit. Med. Journ.*, Dec. 1883.) A number of additional examples were cited in the paper, and in the course of the discussion which followed, but no fresh light was thrown on their nature or causation.

The pathology of these cases is very obscure. The immediate cause of the suppression would appear to be intense congestion of the kidneys. Disturbance of the innervation of the organs is probably the primary cause and possibly, also, in many cases, the direct cause of the suspension of the secretion.

Death is too rapid to be due to the non-elimination of the urinary excreta. The suppression, in fact, is only one among the many phenomena of fatal collapse.

In the way of treatment there is nothing equal to the hot bath. In many cases the relief is both striking and prompt; the flow of urine being sometimes restored while the patient is actually immersed. The effect may be kept up by hot mustard and meal poultices to the loins. Hot gruel enemata are also useful. Medicines by the mouth can rarely be administered on account of the incessant vomiting.

Amongst hysterical patients there is sometimes observed, not merely the common symptom of retention of urine, but true suppression of urine. In many of the cases reported as such, especially of old date, this condition was undoubtedly simulated. Laycock,[2] however, was amongst the first to point out that the suppression may be real. In more recent times Charcot[1] has called particular attention to this fact, and has described a case in which no urine whatever was secreted for as long as eleven days. The utmost precautions were taken to prevent deception. The patient suffered during this time from excessive vomiting, and the vomited matters were found to contain a quantity of urea. No other ill-effects were observed, and the urinary flow was afterwards spontaneously reëstablished.

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[1] *See* a paper, by W. M. Banks, on Urethral Fever, in Edin. Med. Journ. for June, 1871.

[2] Nervous Diseases of Women.

2. *Obstructive Suppression.*

The most common case of obstructive suppression is due to the impaction of a stone in the ureter of a person who has only one kidney, or, at least, only one capable of secreting urine. Sometimes one of the kidneys is congenitally absent; or one kidney has been permanently disabled at some preceding period of life by the lodgement of a stone in its ureter, or by some other accident or disease. In such a case the passage of a stone into the ureter of the surviving kidney would, of necessity, produce complete suppression of urine. The next most common case is due to the blocking up of the terminal portions of the ureters by the progress of a morbid growth, involving the base of the bladder. The less frequent cases depend on some congenital malformation of the ureters, or of the renal arteries, whereby an impediment is constituted to the outflow of urine. This may be slight at first, but in process of time it becomes progressively greater, until at length it arrests the secretion of urine. Examples of these three modes of obstruction will be found among the following cases.[2]

A case of suppression from obstruction seldom reaches a fatal climax without some urine having been voided during its course—it may be a few ounces, or it may be a few pints. The character of this urine is very remarkable. Instead of being high-colored and concentrated—as one would expect under such circumstances—it is pale and watery, and of very low specific gravity. It may accidentally be colored by blood, but it is defective in the proper urinary pigment, and, as a rule, is free from albumen.

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[1] Diseases of the Nervous System, New Syd. Society, vol. i.

[2] [An unusual cause of obstructive suppression of urine recently came under my notice in the Post-mortem Theatre of the Manchester Royal Infirmary. The right kidney was transformed into a large sac of cheesy matter, and similar material filled and completely blocked the right ureter. The left kidney was enlarged, somewhat congested, and contained numerous small masses of tuberculous material, situated for the most part in the cortical portion. The left ureter was slightly distended by clear urine as far as its entrance into the walls of the bladder. The bladder itself was congenitally small, and measured only 1½ inch in length. Its mucous membrane was greatly swollen and congested, and contained numerous tubercular masses. The orifice of the left ureter was surrounded by thick fibrous tissue, and its lumen completely closed by the swollen mucous membrane of the bladder. The suppression of urine had lasted for five days before death. R. M.]

This peculiarity depends on the physical conditions under which the urine in these circumstances is secreted. In order to understand the matter clearly, it will be necessary to call to mind the mutual relations in health of the blood circulating in the renal arteries and the urine newly secreted from it, and flowing down the uriniferous canals. In the normal state, the limiting membrane intervening between the blood circulating in the Malpighian tufts and around the convoluted tubes on the one hand, and the urine in the uriniferous canals on the other, is subject on the side of the blood to a considerable pressure, namely, the lateral pressure within the arterial system; while on the other side there is no counter-pressure at all so long as the escape of urine is free.

This inequality of pressure, as was first suggested by Ludwig, and afterwards experimentally proved by Hermann,[1] is a capital factor in the production of the urine. Hermann (operating on animals) found that when the pressure within the renal artery was lowered, the flow of urine was proportionately diminished. He tested this point in two ways. In the first set of experiments he lowered the blood-pressure in the kidney by contracting (by a clamp) the calibre of the renal artery. In the second set he created a counter-pressure in the uriniferous canals by impeding the flow of urine by means of a column of mercury communicating with the ureter. By this latter method he exactly imitated the condition produced when the ureter is blocked up by a stone, or some other mechanical obstruction. Hermann found that a pressure in the ureter of 10 millimetres of mercury (0.4 inch) caused a sensible diminution in the flow of urine; this diminution went on increasing up to a pressure of 50 millimetres; and with a pressure of 60 millimetres of mercury (2.4 inches) the secretion of urine was altogether arrested. In these experiments the specific gravity and coloration of the urine are not alluded to, but it was uniformly found that the percentage of urea progressively diminished as the pressure in the ureter increased.

Basing our deductions on the clinical facts to be presently adduced, and on the results obtained experimentally by Hermann, we may assume that a mechanical obstruction in the ureter will inevitably produce the following series of events: As soon as the obstruction is established the urine begins to accumulate above it; the accumulating urine determines an upward pressure first in the ureter, then in the pelvis of the kidney, and ultimately in the uriniferous tubes. As the urine goes on accumulating, the pressure within these channels necessarily increases, until at length the pressure so created is sufficiently great in the uriniferous canals to counterpoise the pressure within the renal bloodvessels. When this point is reached the secretion of urine is arrested and total suppression ensues.[1] If, again, the obstruction be not altogether complete, and there be room for some urine to escape past the obstacle, the urine so escaping will have been secreted under a high pressure within the uriniferous canals, and its constitution will be found thereby materially altered; it will be very pale, watery, devoid of its proper coloring matter, poor in urea, and of low specific gravity. It may, indeed, be tinged with blood, but this is an accidental circumstance.

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[1] Henlé and Pfeufer's "Zeitschrift," 1862, p. 1.

Another point with regard to the urine in obstructive suppression is the irregularity of its times of emission. In nearly all the cases observed this is a marked peculiarity. One day there will be a discharge of urine, the next day none, or perhaps none for two or more days, and then again a return of the flow, and again an arrest. This point will be again adverted to.

The long delay of characteristic symptoms is also a striking circumstance. When even the suppression is absolute, seven or eight days elapse before the special symptoms of uræmic poisoning make their appearance, but when these do appear the end approaches rapidly, and death is not delayed beyond two or three days. Up to the rise of the proper uræmic symptoms the condition of the patient is, as a rule, wonderfully calm and free from distress. There may be more or less gastric disturbance and insomnia, and declension of the muscular strength, but the functions generally proceed tranquilly, and the intelligence is undisturbed.

The most distinctive and invariable of the special uræmic signs are muscular twitchings. I believe that these are never wanting. Contraction of the pupils is also a constant sign, but later in its development than muscular twitches. Rapidly increasing muscular weakness is also constantly witnessed, and as this invades the respiratory muscles the breathing becomes markedly slow, panting, and laborious. The tongue and palate become quite dry in the last two or three days. The cerebral functions are much less involved than might be expected.

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[1] In *retention* of urine the obstruction is situated below the bladder—in the urethra; and the physical conditions are essentially different from those of an obstruction in the ureter—on account of the enormous distensibility of the bladder which permits the urine to accumulate in that viscus, and thus prevents the pressure extending immediately up the ureters. But this distensibility is not, of course, unlimited, and a time must arrive—if life continue and the obstruction be not overcome—when the effects of the block are felt in the ureters, and then *suppression* of urine is superadded to retention.

There is increasing drowsiness, with short, fitful snatches of sleep, and a little rambling delirium, but absolute coma rarely supervenes, and convulsions are quite exceptional. The intellect is more commonly preserved to the last, and in more than one instance the patient has spoken sensibly the instant before death. Diarrhœa (unless produced artificially) is quite exceptional, so likewise is excessive vomiting. There is not any dropsical symptom.[1] The skin is commonly moist, often sweating profusely. There is never any ammoniacal or urinous odor from the breath or skin, nor from the body after death.[2] The power of taking food varies: as a rule, it is moderate up to an advanced stage, but complete anorexia comes on a day or two before death.

There are some other points relating to the morbid anatomy, the survivorship, and the treatment, which will be more conveniently noticed in the way of comment on the particular cases to be now described, or in the concluding part of the section.

Case 1.—A man, æt. 67, who twelve years before had suffered from symptoms of renal colic, but had not passed any stone, was attacked about six weeks before his death with symptoms of left renal calculus, with frequent micturition and pains in the left loin, etc. A fortnight before his death, after a long walk, he felt a sudden access of intense pain in the left loin. This continued in great severity for four days, and was accompanied with very frequent and scanty micturition. At the end of these four days the urine became altogether suppressed, and the pain ceased a few hours after. On the third day of complete suppression, I saw the patient. He had absolutely no symptoms referrible to the suspended urinary function; he was calm, free from pain, also from nausea and vomiting, without desire to void urine; pulse 80; tongue clean; skin dry; he had had no sleep for two nights. He was ordered a warm bath, a saline mixture, and to have the course of the left ureter well kneaded with the aid of a liniment. Next day (fourth day of suppression) he passed a pint of pale, limpid urine; he had perspired freely and slept some hours. On palpating the renal regions, the right was felt to be flat and empty, contrasting with the left, which presented its natural roundness and sense of resistance.

On the next day (fifth) twelve ounces of urine were voided. It was clear, almost colorless, sp. gr. 1010, not albuminous, and contained 1.92 grain of urea per ounce. There were anorexia, thirst, nausea, and occasional vomiting, a slight sense of mental confusion, but no actual delirium; pulse 80; respiration 24.

On the following day (sixth) the same symptoms continued, with intense restlessness and insomnia. Sixteen ounces of colorless urine were passed, sp. gr. 1011, containing 2.08 grains of urea per ounce. The following new symptoms also showed themselves—dryness of tongue at tip, contraction of pupils, and occasional hiccough. In the evening of this day, six more ounces of limpid urine were voided; sp. gr. 1011; temperature in axilla 98.6°.

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[1] For an exception to this rule, *see* Case 10, further on, and Dr. Russel's Case in Med. Times and Gaz., 1879, I. p. 474.

[2] This seems a point of distinction from *retention* of urine.

On the afternoon of the next day (seventh) a great change for the worse was observed. Pulse 80, irregular; respiration 20, labored, long-drawn, interrupted; tongue dry and brown; frequent muscular twitches all over body; patient indifferent and drowsy, but answering questions intelligently; no urine for the last eighteen hours.

Death took place thirty-six hours after the last visit—exactly nine and a half days from the commencement of the suppression. The symptoms during this last period, as observed by the late Mr. Mellor, with whom I saw the case, were—increased laboriousness and slowness of the respiration, which assumed a panting character; deepening indifference, but still he answered "yes" and "no" to questions addressed to him, though slowly and unwillingly; pupils contracted to pin's points; finally complete coma. There was a doubtful convulsive seizure immediately before death.

Autopsy.—Strong rigor mortis; body well nourished, and quite free from urinous or ammoniacal odor. All the organs healthy except the kidneys and ureters. The *right kidney* was wholly converted into a fibrous mass, studded with cysts, and weighed two and a half ounces. The corresponding ureter was impervious throughout, and changed into a fibrous cord, which was thickened about the middle to double its width. This thickened part was solid and fibrous like the rest. No stone existed in any part of the ureter or kidney, but it was conjectured that the thickened part of the ureter had been the seat of an obstruction, and that the stone, or whatever object had constituted the obstruction, had been subsequently removed by absorption.

The *left kidney* was much enlarged, it weighed ten ounces, and, on section, appeared dark and intensely congested. The ureter was as thick as a goose-quill, and distended with urine. At its lower part were found three little oxalate of lime calculi about the size of hemp-seeds, and weighing altogether a grain and a half. One of these was tightly impacted in the terminal part of the ureter, where it passes through the coats of the bladder; this was the cause of the obstruction. The fluid imprisoned in the ureter amounted to three drachms, and consisted of grumous bloody urine. The pelvis of the kidney was only slightly dilated, and contained about two drachms of bloody urine.

The bladder contained about six ounces of pale dilute urine; its coats were healthy.

The course of events in this man appears to have been the following: About a month before the patient came under observation, three small calculi, which had been previously lying harmlessly in one of the infundibula, were dislodged, and fell into the pelvis of the kidney. Here they sojourned some four weeks, causing pains in the left loin and frequent micturition. At the end of this period they suddenly entered the ureter, and for four days, amid great suffering, continued their descent to its lower part. Here the foremost calculus became impacted, the pain ceased suddenly, the passage of urine was blocked up, and suppression ensued. Had the opposite kidney been intact, no serious consequences would have followed. The healthy kidney would have become proportionately hypertrophied and performed double duty. But the right kidney was, by an untoward coincidence, practically non-existent. It had itself, as may be conjectured, many years before passed through a train of events similar to that which had now extinguished the activity of its fellow.

The suppression of urine in this case lasted nine days and a half. During the first three days the suppression was complete. Then followed a period of four days, during which an aggregate quantity of fifty-four ounces of urine was voided. Finally, in the last two and a half days no urine was passed, but six ounces were found in the bladder after death, making a total of sixty ounces of urine secreted in nine days and a half. This seems at first sight a not inconsiderable quantity, and causes surprise that, suppression being so incomplete, life was not longer maintained. But on closer inquiry the suppression proves to have been more complete than at first appeared. The urine discharged was exceedingly dilute, its sp. gr. ranged from 1010 to 1011, and its proportion of urea was only about two grains per ounce; this gives a total weight of urea excreted in nine and a half days of only 120 grains, which is less than one-fourth of the normal amount for a single day.

CASE 2.—A very stout, tall man, æt. 59, suffered four years before from symptoms of the passage of calculi from the left kidney. Two small uric acid stones were passed after several weeks of suffering, and then the symptoms subsided.

After four years of good health, the patient was seized one morning, without assignable cause, with sudden pain in the right loin and urgent desire to pass water. The pain and urgency of micturition continued until the afternoon, and small quantities of bloody urine, amounting altogether to about half a pint, were voided at short intervals during the day. The stomach was irritable throughout the day. Toward evening the flow of urine ceased entirely, and the pain diminished.

I saw the patient for the first time about fifty hours after the commencement of the suppression, with Mr. Grindrod, of New Mills; and I visited him daily until his death, which took place nine days and a few hours after the arrest of the urinary flow. During this period he only voided urine once, namely, two ounces on the fourth day, and none was found in the bladder after death. This specimen of urine was quite characteristic of obstructive suppression. Its sp. gr. was 1010; it contained a little blood, and a slight corresponding trace of albumen. When the blood-corpuscles had subsided, the urine had a pale straw color, and the deposit contained, besides blood-disks, a large number of epithelial cells of a transitional character, resembling those of the pelvis of the kidney.

The case, which was closely watched throughout its course, presented a typical example of death from pure anuria. Dr. Garrod was telegraphed for from London, and joined our consultation on the fifth day of suppression.

For the first six days the symptoms were marvellously slight, and yielded but faint indications that one of the capital functions of the body was in absolute abeyance. The muscular strength had indeed declined, and the sleep was bad, but the patient was calm; his tongue, skin, and pupils were natural; there was little nausea, and no vomiting after the fourth day; the intellect was unclouded; there was not the least urinous or ammoniacal odor about the breath or sweat; the pulse was steady, at about 72, the respirations 24, and the temperature scarcely varied from the normal limits. There was no desire to make water, scarcely any pain or tenderness in the right loin, and he continued to take a fair amount of nourishment. On the seventh day the characteristic symptoms of suppression began to show themselves. On this day occasional slight twitchings or pluckings of the muscles were observed on the trunk and limbs, and the tongue began to be dry. The insomnia, which had been a marked symptom from the first, became very distressing; he dozed frequently for short periods, and started on falling asleep and awaking. He took nourishment fairly, and had no vomiting or thirst, and only very slight and transient nausea.

On the eighth day the patient was still calm, and quite free from mental confusion or indifference when fairly awake, but when left alone he was constantly falling off in a fitful doze, and awaking with a start. The muscular twitchings were more marked than yesterday, and the muscular weakness had increased greatly; nevertheless, he was up and dressed in his bedroom for an hour and a half. The pupils were natural, and he took his food pretty well—a quart of milk, some cocoa, bread and butter, and rice pudding. The skin had acted profusely from the beginning in response to warm baths. No nausea or vomiting. A peculiar panting character of the respiration was noticed to-day, which became more and more pronounced until his death. The temperature also began to fall.

On the ninth day the patient's condition changed greatly for the worse. The insomnia and restlessness were most distressing; the twitchings of the muscles very frequent and severe; the tongue and mouth were perfectly dry; the pupils were decidedly contracted, though still sluggishly responsive to light; thirst was troublesome, and the appetite quite gone; the weakness was so great that he could not walk without the help of two assistants; his legs had to be lifted into the bath. There was no persistent nausea, but he vomited after a compound jalap powder. Although his intellect was clear when he was roused (he transacted some business with his lawyer), there was marked indifference when he was left undisturbed, and he lapsed at once into a dozy state, lying with his mouth open and jaw half dropped, breathing pantingly with long pause between expiration and inspiration.

On the tenth day, at 1 P.M., the patient died, having lived for a little more than nine whole days from the onset of the suppression, and having voided in this interval only two ounces of a very dilute urine.

The incidents of the closing scene were very distressing. The weakness increased rapidly; the night was most restless; the patient was constantly getting up to have a stool, but voided nothing except a little mucus. The thirst, dryness of the mouth, and the muscular twitchings went on increasing. At 6 A.M. the breathing became very embarrassed, threatening suffocation. He asked to be instantly raised on the side of the bed into a sitting posture. He then belched up a large quantity of flatus, and was thereby much relieved in his breathing. After a couple of hours he lay down again, but with his head raised. The power of his legs was now quite gone; he said he could not feel them. At nine o'clock the pulse was 80, respirations 15, very labored and interrupted. The pupils were strongly contracted. The twitchings were incessant all over the body and limbs. The breathing becoming again more embarrassed, he was lifted on the side of the bed, and finally into his armchair. His strength failed now more and more, and the breathing became more and more difficult, and the uneasiness and distress increased, dozing and starting incessantly. He remained in his chair until one o'clock, when he began to slide off, and, while about to be assisted up again, he asked to have his hands rubbed, and suddenly fell back dead. There was neither coma nor convulsions throughout. He appeared to wander at times through the night, but when his attention was roused, he showed unshaken consciousness and intelligence to the end. The character of his breathing in the last few days was peculiar, and became increasingly so as death approached. The inspiration became more and more prolonged and laborious, and expiration shorter and more panting, with a lengthening pause between. The respiratory difficulty, which appeared to be the immediate cause of death, evidently arose from the diminishing power of the inspiratory muscles.

The *post-mortem* examination was confined to the abdomen. All the organs were healthy, except the kidneys and ureters. The *right kidney* was enlarged, and weighed 11½ ounces. Its surface was dotted here and there with numerous black blood-spots; but the general appearance, both on the surface and on section, was pale mottled, decidedly anæmic-looking. It contrasted strongly with the dark, almost black congested kidney found in Case 1. The pelvis and ureter were not in the least dilated. They contained about two teaspoonfuls of blood-stained urine. A small uric acid calculus was found tightly impacted in the lower part of the ureter, just above its entrance into the bladder. It was about the size and shape of a hemp-seed, and weighed 1⅓ grain.

The left kidney was found completely destroyed. It was hollowed out into a lobulated sac, about as large as the healthy kidney. On cutting it open, there escaped about five ounces of an opaque white fluid, exactly resembling new milk. This singular-looking fluid retained its milky appearance, even on long standing; it was found to consist of myriads of needles of urate of soda floating in a highly albuminous serum. The sac wall consisted of a tough leathery tissue, from one to two lines in thickness, quite devoid of any recognizable renal structure. The cause of this mischief was found at the entrance into the ureter, where the channel was completely blocked up by a uric acid stone, weighing 52 grains. The rest of the ureter was pervious and normal.

The bladder was empty and healthy. The body generally was perfectly sweet and free from any urinous or ammoniacal odor.

The pathological story of this man's case was easily read even during life, and only a few details were left to be filled in at the autopsy. The left kidney was destroyed four years before by the impaction of a calculus in its ureter. The right kidney then became hypertrophied, and performed double duty in a perfect manner until another calculus blocked up the right ureter. Then the secretion of urine was suddenly and permanently arrested, and the patient destroyed in less than ten days.

In reviewing the symptoms in this case, it may be observed that insomnia and progressive failure of the muscular strength marked the entire course of the case. A certain disturbance of the stomach and slight febrile movement set in when the stone was impacted in the ureter; but these passed away after the fourth day. A fair amount of nourishment was taken up to the eighth day, after which the power of taking food almost wholly failed. The movements of the pulse, respiration, and temperature may be seen by a glance at the following table:

	Pulse.	Respiration.	Temperature.
Third day	72	—	—
Fourth day	72	24	100.0°
Fifth day	72	24	99.7
Sixth day	72	24	99.7
Seventh day	76	20	98.6
Eighth day	76	22	98.2
Ninth day	76	20	97.4
Tenth day	80	15	—

The pulse remained almost stationary, but with a slight tendency to increased frequency. The respiration showed a tendency to diminished frequency, especially toward the last. The temperature manifested a steady tendency to diminution, especially as death approached. This, I believe, will be found to be the general rule in fatal anuria. Muscular twitches were first noticed on the seventh day. At first they were slight and infrequent, but they became more and more frequent and severe as the case approached its termination. The faculties were clear to the last gasp; there existed, however, in the last three days a constant tendency to lapse into indifference, with fitful dozing and starting, when the patient was left undisturbed. The pupils did not show decided contraction until the ninth day, and dryness of the tongue and mouth became a marked feature on the same day.

This case and Case 1 illustrate a noteworthy point in the morbid anatomy of obstructive suppression. In both of them it is noted that the ureter above the obstruction, and the pelvis of the kidney, although moderately filled with stagnant urine, were not materially dilated or enlarged. Those examples of monstrously enlarged ureter and pelvis (sacculated kidney or hydronephrosis), which are often witnessed as the effects of obstruction in the ureter, are produced by slow degrees, and must be regarded as a growth rather than a simple dilatation. Indeed, the ureter and renal pelvis appear incapable of that rapid dilatation which we are familiar with in the bladder. This consideration enables us to explain how two different results may follow one and the same cause, namely, obstruction in the ureter. When the obstruction is suddenly established and is at once complete, the consequence is not enlargement and sacculation, but atrophy of the kidney and ureter. When, on the other hand, it is slowly established and is incomplete, it produces hypertrophic dilatation of the ureter and pelvis, and eventually sacculation of the kidney or hydronephrosis.

CASE 3.—A man, æt. 40, had suffered three months before from symptoms of renal colic on the *right* side, and voided some small calculi. He soon recovered from this attack, and went about his business in his usual health, until three weeks before his death. He then began to suffer from pain in his *left* loin, which continued for a fortnight. During this period the urine was voided in apparently the usual quantity, but his wife noted that it had entirely changed its character. Before it had been high-colored, but now it became "clear as water." At the end of the fortnight complete suppression of urine came on, and death ensued in five days.

I only saw this man once, on the day before his death, in consultation with Mr. Edwards, of this town. He was then in a state of full uræmic intoxication—pupils contracted to pins' points—muscular twitchings universal over the whole body—breathing panting, slow, and interrupted—tongue and mouth quite dry. He was very restless, and almost indifferent, yet he answered questions sensibly when roused. He died next day without coma or convulsions; he spoke sensibly half an hour before his death.

Autopsy next day. The body was quite free from urinous or ammoniacal odor, and healthy in every part except the urinary organs.

The *right kidney*, which was about the normal size, was hollowed, and in process of atrophy; the cortical substance alone partially remained, and this was pale and wasted. The infundibula were moderately distended, and contained about an ounce of pale fluid, which was lost. The right ureter was plugged up at its commencement by an elongated uric acid stone, weighing twenty-two and a half grains. Another little stone, as big as a hemp-seed, lay in one of the infundibula. The ureter below the plug was normal.

The *left kidney* was much enlarged, but healthy. It had the mottled appearance of the right kidney in Case 2. Three little uric acid calculi, like flattened mustard seeds, lay free in the infundibula. The ureter and pelvis were moderately distended with fluid; the ureter appearing about the size of a crow-quill. On slitting it open, superficial abrasions were seen along its entire track, showing the footsteps of a descending calculus. Near the bladder this calculus was found, at the termination of the ureter. It slipped into the bladder during the manipulations. It was a round uric acid stone as large as a small pea, and weighed a grain and a half.

The bladder was empty and healthy.

Though this case was seen but once, the diagnosis presented no difficulties. The course of events was evidently as follows: Three months before the fatal attack the right ureter was plugged by a calculus, the function of the right kidney was thereby permanently extinguished, and the organ at once passed on to a state of atrophy, which was nearly complete at the time of death. The left kidney then took up the double duty, and became proportionately hypertrophied. The calculous tendency, however, was not arrested, and about three weeks before death a small calculus passed into the left ureter. It continued to descend amid much suffering, for about a fortnight, causing partial suppression of urine. The urine voided during this period had the special characteristic of urine secreted under pressure from below—*i. e.*, it was pale and watery. At the end of the fortnight the calculus had reached the terminal portion of the ureter; there it became immovably impacted, complete suppression ensued, and death followed in five days. It must be assumed in this case that during the fortnight of partial suppression a certain degree of blood-poisoning took place from the accumulation in the blood of the effete ingredients which should have been removed by the kidneys, so that when the suppression became complete it only required five days (instead of nine or ten) to render the blood poisoned to such a degree as to be incompatible with the maintenance of life.

CASE 4.—A man, æt. 65, had been subject for some years to attacks of renal colic, and had from time to time voided uric acid calculi. Some fourteen days before my visit, symptoms of left renal colic had set in, with pain in the loin and frequent micturition. I was informed that during these fourteen days a considerable quantity of pale, clear urine had been voided, averaging altogether about two pints a day, but discharged irregularly. On some days none had been discharged, while on other days it had flowed copiously at two or three separate micturitions.

When I saw the patient he was in the last phase of uræmia; the pupils were strongly contracted; there were frequent and universal muscular twitchings; pulse 100; respirations 16, markedly panting, but consciousness was intact when the attention was roused.

The hypogastrium being protuberant and dull, a catheter was introduced, and two pints of urine were withdrawn. This presented the usual characteristics of obstructive suppression, it was very pale, and its sp. gr. 1006.

Death took place on the fifteenth day of suppression, which, however, had only been partial throughout. A post-mortem examination was not permitted, but it was not difficult to divine what had occurred. The right kidney had doubtless been destroyed at some previous period by

the impaction of a calculus in its ureter. The left kidney, which had then become the sole organ of the urinary function, was in its turn subjected to a similar accident; a calculus entered its ureter and failed to clear the passage into the bladder, incomplete suppression ensued, and death in fifteen days.

This case is instructive in one respect, and suggestive of a caution in judging of the amount of urinary secretion. This man voided on an average about two pints of urine daily. Had this amount been of normal density and appearance, it would have indicated a degree of renal activity certainly equal to the prevention of uræmic poisoning. Patients may live for months without voiding more than fifteen or twenty ounces of urine a day, as is frequently witnessed in cases of cirrhosis of the liver and in regurgitant heart disease. But in these cases the urine is always of high density, deeply colored, and fully charged with urinary ingredients. Here, on the contrary, the urine was pale and dilute, and the density of the specimen examined was only 1006. What amount of normal urine this represented cannot be accurately determined, but judging by the result of my analysis of the urine passed under similar circumstances in Case 1, the urea would not amount to more than about one grain to the ounce. Calculating on this basis, this man excreted only forty grains of urea per day, which is not more than one-tenth of the normal amount. Another point in the case deserves notice as being more or less constantly characteristic of the mode of emission of urine in obstructive suppression; this was the irregularity of the times of discharge. Although the patient in this case discharged an average quantity of two pints a day, this was not voided with that approach to regularity which marks the normal state, but most irregularly; one day no urine at all would be voided, the next day it would be voided copiously two or three times, then again none at all for two or three days, and so forth. I have noticed this paroxysmal character of the urine-discharge in all my cases of obstructive suppression, and I believe it to be a point of considerable diagnostic value.

The two following remarkable cases show that recovery is possible even after very protracted suppression of urine, provided the flow of urine can be reëstablished. The notes of the two cases were furnished to me by Dr. Clifford Allbutt, of Leeds, and Dr. Duigan, of Gainsborough, respectively. In the first case the suppression continued for nearly ten days, and in the second for nine days. In neither case were twitchings of the muscles noted, but the pupils had become contracted in Dr. Allbutt's case, and there was some mental confusion. From my own experience I should regard muscular twitchings as *the* first really undoubted and characteristic symptom of uræmic poisoning; it cannot, therefore, be said that recovery followed in either case after the full declaration of uræmic symptoms. Another apparently well-authenticated case of recovery after nearly ten days' total suppression, of obscure nature, is recorded in the tenth volume of the *Edinburgh Medical and Surgical Journal*, p. 409.

Case 5. (From the notes of Dr. Allbutt.)—Mr. W., a healthy vigorous man of about 56, was first seen by Mr. Wheelhouse, on Wednesday, September 11, 1867. He complained of great lumbar pain, weight, sense of fulness, sickness, and febrile disturbance.

Monday, 16th.—Symptoms of descent of calculus along ureter commenced.

Saturday, 21st.—During this time stone apparently traced along ureter.

October 2d.—Stone from last date till now seemed to be impacted at entrance into bladder, constant pain augmented in paroxysms till 3 A.M. this morning, when sudden and entire relief was felt, and the patient was told how to look for symptoms of stone in the bladder. At 6 A.M. he passed the last quantity of urine, about ℥ij. Up to this time the flow had been free and the fluid normal.

3d, 9 A.M.—No urine passed. Catheter used, but no obstruction found. Bladder quite empty. 3 P.M.—Same state. Perfect freedom from pain, no urine. No symptoms of uræmia. 10 P.M.—Consultation with Dr. Allbutt. Same state. Temperature 100°. Hot bath and fomentations ordered.

4th (Friday), 9.30 A.M.—Same state. No urine. No uræmia. Much local uneasiness and restlessness. Temperature 98.2°. Fomentations, saline purgatives and diluents. Bromide of potassium with a little iodide given as a sedative, opium being inappropriate. 9 P.M.—Same state. A drop or two of urine had been coaxed out, just enough to make a stain at the bottom of a small vessel. No symptoms of poisoning. Patient quite clear and much more comfortable.

5th.—Mr. W. summoned at 5 A.M. Much pain at the old point; cramped limb of same side; not a drop of urine, though frequent solicitations; firm pressure on part gives relief. Sp. æth. sulph. ordered every half hour. 8.30.—Seen with Dr. Allbutt. Pain subsided after a few doses of ether; no urine; breath sweet; perspiration normal. On examination whole left side of belly from middle line dull; left rectus tense; dulness varies a little with position. Patient clear and intelligent; no drowsiness. Ether and bromide omitted. 3. P.M.—Same condition; pain returning; no urine; no uræmia. 9.30.—Seen with Dr. Allbutt. Physical examination: Dulness over whole of hypogastrium below a cross line drawn through the navel; dulness little affected by position. Examination per rectum showed only a tender spot behind the prostate; no bulging; catheter passes freely, and is moistened with a few drops of urine, perhaps twenty or thirty drops; upon the end of it is a little bloody mucus. Breath decidedly urinous;

mind clear; no headache. Pulse weaker and quickening a little. Pulse and temperature have been normal.

6th, 9.30.—Pulse 96, better; temperature 98.2°. Had passed a fair night; no urine. Dulness of belly extends a little above navel on left side, but not extending so far to the right as yesterday. Breath *not* urinous. Bowels have been kept open by salts until to-day, when no motion was reported. 9.30 P.M.—Singularly clear in head; placid sleep for five hours. Two watery stools. No urine, unless it be a very few drops passed after repeated efforts; is cheerful, and walks about the room easily, and is well able to sit down and rise. A little cough which he has seems to shake and hurt the lower belly. Tongue coated, but food taken fairly in small quantities. Has had, for instance, a little partridge to-day. Pulse and temperature normal. Breath sweet. Ankles not puffy. Dulness all over hypogastrium.

7th, 9.30 A.M.—Good night. Pulse natural. Temperature 97°. No stupor or headache. Sense of a movable tumor in lower abdomen. A few drops of urine, perhaps a teaspoonful, accumulated after repeated efforts. 10 P.M.—Complains of weight at lower belly on left side, and pain there on coughing. Sickly during the day. Pulse and temperature normal. No uræmic symptoms.

8th, 9th, and 10th.—Same report, unless there be a little drowsiness and tendency to be a little "lost" at times.

11th.—This morning a little urine was passed, quantity not recorded. There is a good deal of mental oppression, especially after awaking. Aspect dull and heavy. Pupils contracted. Dulness of abdomen about the same; it is a little increased on left side, but diminished a little to the right. He has been purged to-day without medicine.

12th.—Has passed ℥ivss of water, and there is a little less mental obfuscation. Has had a warm bath, which relieved him in every way. Is still purged also, an action which is not prevented. Tongue loaded, appetite nil. Temperature normal.

13th.—Marked improvement; a copious flow of urine last night. The head clear; a refreshing night. Some return of appetite. Abnormal dulness much diminished.

14th and 15th.—A good deal of pain, dragging and paroxysmal; chiefly in the old place, above and to left of pubis; is irritable and restless; expression worn and anxious. There is no pain at the end of the penis. Pulse 100, weak. Temperature 100°. As the water is now very abundant, we are able to give him champagne and morphia injections, which with warm water baths relieve him. Is still purged.

16th and 17th.—Pains cease. No stone is discovered. Convalescence.

21st.—May be considered well. Functions normal. Appetite good. No dulness in abdomen.

I strongly suspect that the suppression in this man was not due to the impaction of a calculus in the ureter, as seems to have been the impression of Mr. Wheelhouse and Dr. Allbutt, but to the existence of a double hydronephrosis, and that the case was similar, pathologically, to one which fell under my notice some three years ago, and which will be related hereafter (see hydronephrosis, case of J. S.). Temporary suppression of urine, extending over some days, followed by copious flow of urine, is a distinctive feature of cases of hydronephrosis; and the extensive dulness in the abdomen, which disappeared after the urine began to flow, can (the bladder being emptied) scarcely be otherwise explained.

In the next case, however, the suppression was undoubtedly due to the impaction of calculi in the ureter, and ceased when these were voided.

CASE 6. (From the notes of Dr. Duigan.)—The patient was a strong, stout, middle-aged cattle-jobber, living in the country. He had often suffered from renal colic, and had frequently passed uric acid calculi. The attack began with pain in both loins, and the patient had had complete suppression for three or four days when first seen by Dr. Duigan, in consultation with Dr. Smallman, of Willingham. The pain had then completely subsided, and except for loss of appetite and the suppression, the man presented no marked symptoms. The introduction of a catheter showed that the bladder was empty. For nine days he continued in this state, never passing any urine all that time, and not suffering from any bad symptoms, sickness, or other indications of uræmic poisoning. At the end of this period the kidneys began to act, and he passed a quantity of clear urine of low specific gravity, containing nothing abnormal. With this urine he voided three or four uric acid calculi, and shortly after got quite well.

In this case it is probable that one kidney had been destroyed at some former period by the impaction of a calculus in its ureter; at the same time it is not absolutely impossible, as Dr. Duigan suggests, though, I think, highly improbable, that both kidneys may have been sound, and that both ureters were obstructed by calculi at the same moment.

CASE 7.—A man, æt. 59, was visited by me with Dr. Herbert Renshaw, of Sale, on July 10, 1871. Six months before he began to suffer from pain in his back, loss of appetite, failure of strength, and constipated bowels. The pain in the back was of a constant and severe aching character, requiring endermic injection of morphia for its relief. The urine was pale and abundant, but discharged irregularly. It did not at any time up to my visit contain blood or albumen.

A month before, the patient had total suppression of urine for four days. This was overcome by compulsorily walking him about between two assistants. The urine returned and the pain subsided. After this, however, the discharge of urine was extremely irregular, and it was noticed that when the urine flowed freely the pain in the back was relieved, and that the pain became aggravated when the urine was for a time suppressed.

After the above-mentioned four days' suppression, he recovered a good deal, and went to Southport. There he was attacked with diarrhœa, and had to return home in consequence.

At the date of my visit he was suffering severely from the pain in the back; he was very weak, and his legs were slightly œdematous. He was then passing from one to two pints of a dilute urine daily; this contained a trace of albumen. I requested that all the urine which the patient voided should be collected and brought to me day by day for the next three days. The first day he voided two pints, the second day one pint, and the third day eight ounces. For the next three days the urine was totally suppressed, and he died. The specimens of urine were all alike; they were pale and watery, the specific gravity ranged from 1009 to 1010; they were acid, and contained a trace of albumen.

The symptoms during the last three days of life were as follows, according to the statements of Dr. Renshaw and the patient's wife, for I saw him only once myself: Increased weakness, marked panting breathing; diarrhœa for the last two days; twitchings of the muscles; rambling delirium when left to himself, but perfect consciousness to the last when his attention was roused; no coma, no convulsions.

Autopsy.—Body quite free from urinous or ammoniacal odor. All the organs were healthy except the urinary apparatus. The source of mischief was found to be a hard scirrhous mass, as large as an orange, which half filled the pelvis. This growth involved the base of the bladder and the prostate gland. The rectum was adherent to it and constricted for the space of an inch; but I could get two fingers through the narrowest part. The seat of the scirrhous growth in the bladder was the submucous tissue. Neither the mucous nor peritoneal coats were implicated, though much puckered and folded, owing to the contraction of the thickened wall of the bladder. The whole trigone was involved, and the disease extended for a full inch above the trigone, terminating in a thick, abrupt rim or border. The walls of the bladder in the implicated region measured from half to three-quarters of an inch in thickness. The fundus of the bladder was quite healthy, and the organ was capable of containing about half a pint of urine. The urethra for the length of an inch passed through the dense mass of the prostate, which was fully an inch and a half thick. The channel was quite free, a catheter had been repeatedly passed during life without any difficulty.

The terminal portions of both ureters passed for the length of an inch through the scirrhous mass; their course in this part was tortuous, and their channel compressed by the surrounding growth, but a probe could be insinuated through both of them, showing that neither was completely occluded. Above the bladder both ureters were dilated to the size of the little finger (the left more than the right), and distended with urine. The *left kidney* was greatly atrophied, and weighed only 2½ oz.; the interior was hollowed, without trace of pyramids, and the cortical substance was reduced to a fleshy rim of tissue of homogeneous appearance. The *right kidney* was enlarged, and weighed 7 oz.; it was hollowed, but not so completely as its fellow. The pyramids were gone, and the cortical substance was undergoing absorption. The pelvis was enlarged to the size of an egg, and distended with urine.

It was evident that the left kidney had not done any duty for some months, and that life had been sustained by the hypertrophied right kidney until its ureter also was blocked up by the progress of the growth in the bladder.

The tumor had contracted adhesions to, and made extensions into, the adjacent parts in the pelvis. The iliac vessels passed through a dense scirrhous mass, whereby they must have been more or less compressed; this was evidently the cause of the œdema of the legs.

My notes of the next two cases are imperfect, but as each of them illustrates some point in the history of obstructive suppression, I will add them to the series.

Case 8.—This was an old lady of about 60, whom I saw with Dr. Gardiner, of Ashton. She was afflicted with cancerous disease of the uterus and vagina, involving the base of the bladder and (presumably) implicating the terminal portions of the ureters. When I visited her no urine had been passed for four days, and the suppression continued without interruption for three days longer, altogether a total of seven days. After this the urine returned, and flowed normally for the remaining four weeks during which she lived. During the time of suppression there were great restlessness and insomnia, with a flushed and anxious expression of countenance, but no twitchings of the muscles, and no convulsions nor coma. There was no autopsy.

Seven days of suppression of urine, without the development of uræmic symptoms, and issuing in recovery so far as the suppression was concerned, is, as we have seen, not an unprecedented occurrence. It may be conjectured that in this case one ureter was permanently occluded by the morbid growth; and that during the epoch of suppression the opposite ureter had become blocked up, probably by a fungous excrescence projecting into its calibre, and that an ulcerative process at the end of seven days again cleared the passage. This is a process analogous to that which sometimes occurs in scirrhus of the pylorus, when the strictured state prevailing in the earlier periods is afterwards removed by the softening and ulceration of a portion of the cancerous mass.

Case 9.—A man of about 35, greatly given to alcoholic excesses, was seen by me on January 15, 1869. He had then passed no urine for four days. He was somewhat stout, and both loins were doubtfully thought to be the seat of bulging, of an elastic, quasi-fluctuating character. The previous history threw no light whatever on the nature of the case. There were no uræmic symptoms, but a great sense of tension of the abdomen. I saw this man on three successive days, and introduced a tubular needle to the depth of three inches into one of the lateral bulgings, but without reaching any collection of fluid. The notion I entertained was that a double hydronephrosis existed, and that the swellings in the loins were the sacculated kidneys distended with urine. He died two days after my last visit. No post-mortem examination was permitted. The suppression lasted nine days, and during that period only about an ounce of urine was voided, which was said to be pale. Up to the seventh day of suppression there were no twitchings of the muscles nor marked contraction of the pupils. The information respecting the final symptoms is defective. There were great restlessness and insomnia. Consciousness was maintained to the last, and the patient asked to be prayed with just before his death.

The following case is remarkable in two respects—namely, the long survival of the patient (15 days) and the occurrence of transient anasarca.

CASE 10.—Mrs. P., æt. 56, had been suffering for 18 months from occasional uterine hemorrhage, due to scirrhus of the os and cervix uteri. On January 15, 1876, the urine became very scanty, and next day it altogether ceased. From this date to the day of her death, on January 30th, not a drop of urine was secreted. I saw her on the tenth day of suppression, with my friend Dr. Lloyd-Roberts, to whom I am indebted for the notes of her case. Her condition was singularly calm. She took her food, and slept well; the tongue was moist and the pupils natural. Pulse 84; temperature 99°. There was no pain anywhere. Dr. Roberts informed me that on the first two days of suppression there was slight general anasarca—most marked in the face. This symptom entirely passed off on the third day; but there was a slight recurrence of œdema in the feet on the day before death.

On making a digital examination, an extensive scirrhous growth was found, involving the cervix uteri and the adjoining part of the vagina corresponding to the base of the bladder. This explained the suppression; the ureters, as they passed through the trigone, were doubtless involved in the cancerous growth, and thereby occluded. The bladder was found empty.

The symptoms continued almost unchanged until January 29th. On that day she became worse. The pupils became contracted; and muscular twitches were observed in the face. The muscular power failed rapidly, first in the arms and legs, and lastly in the trunk. She died quietly on the morning of January 30th, apparently from paralysis of the respiratory muscles. The temperature on the 29th sank one degree below the normal. No autopsy was permitted.

The *duration of life* in complete obstructive suppression appears to range, as a rule, from nine to eleven days, and the passage of a few ounces, or even two or three pints, of a dilute urine does not seem to extend the time of survival beyond a few hours. I have not discovered more than four well-authenticated cases (in addition to the one just related—Case 10) in which suppression of urine was complete, or approached completeness, where the patient survived beyond the eleventh day. The first of these is recorded by Rayer (*Mal. des Reins*, t. iii. p. 490). The patient was a man of 64 years of age, who had hydronephrosis of the right kidney of many years' standing. The ureter of the left kidney was blocked up by a calculus, and suppression of urine ensued. This proved fatal in twenty-five days, and in that interval only two ounces of urine were voided. The second case is described by Sir J. Paget in the second volume of the *Transactions of the Clinical Society*. The patient was seventy-three years of age. The right kidney was atrophied and apparently incapable of secreting any normal urine. The left kidney was hypertrophied and the ureter blocked by a stone. Complete suppression ensued for thirteen days. No symptoms of uræmic poisoning appeared until the last of these thirteen days, when a slight attack of convulsions occurred. Then, on the fourteenth day, he passed an uncertain but "considerable" quantity of urine, and again six ounces on the same day; some slight convulsive movements which had been observed during the day then ceased. From this period until his death, seven days afterwards, the suppression was complete, and no urine was found in the bladder after death. So that there was total suppression for twenty-one days, only interrupted by one day's emission of urine. Muscular twitchings made their appearance on the sixteenth day. Sir J. Paget attributed the extraordinary protraction of life in this case mainly to the patient's advanced age; but this view is scarcely borne out by other experience. My first patient was sixty-seven—only six years younger than Sir J. Paget's case, yet he only survived nine and a half days, though he secreted sixty ounces of urine in that period.

A third case is reported by Dr. Russell in the *Med. Times and Gazette*, 1879, i. p. 474, where the patient, after complete obstructive suppression lasting twenty days, recovered for a time, but died afterwards. There was excessive vomiting and also general œdema. A post-mortem examination showed the presence of calculi in the pelvis of each kidney, blocking the ureter.

Tenneson (*Gaz. Hebdomad.*, 1879) reports a case in which the suppression of urine lasted fifteen days before death ensued. Œdema was also present in this case.

There are, indeed, other cases on record, in the more ancient literature of medicine, in which patients are alleged to have survived many months of total suppression of urine; but it may be safely affirmed that imposition of some sort or other was practised in these cases.

TREATMENT.—Our notions of the treatment must vary according to the nature of the obstruction. Taking first those cases which are due to impaction of a stone in the ureter, it must appear that the use of ordinary diuretics cannot avail against a physical obstacle. Reliquet[1] has recorded a successful case in which he applied gutta-percha bands to the limbs, with a view of increasing the blood pressure in the kidneys. There is something to be said in favor of means directed to excite the contractile power of the ureter. In my second case Dr. Garrod suggested, with this view, the use of turpentine, but it provoked vomiting and could not be persevered with. In a case reported by Carrère (*Gaz. Hebd.*, 1879) ergot was given, recovery following shortly afterwards. Or remedies of an opposite class might be alternately tried with the purpose of relaxing the spasm of the ureter, such as opium, chloroform, belladonna, venesection, and warm baths. My own impression, however, is more in favor of mechanical means; and in reviewing the cases which have fallen under my notice, I cannot help thinking that something further might have been attempted in this way with a prospect of advantage. One such means, namely, kneading and shampooing the renal region and the course of the ureter, was in two of my cases followed by a so immediate, though only transient, flow of urine, that I could scarcely doubt that it was due to the means employed.[1] But in a large number, if not the majority of cases the impaction takes place near the bladder, where no direct force can be applied. Indirect means may, however, be tried. The physical condition is generally this: Above the calculus the ureter is open and distended with stagnant urine; at the seat of the lodgement, and below it, the ureter is contracted. A displacement either upwards or downwards would be likely to be followed by relief. To provoke such displacement, succussion of the body and various changes of posture might be tried. The patient should be directed to support himself from time to time on his knees, with the upper half of the body depressed, and the sacrum might be repeatedly struck with the fist. The force of gravity would thus be brought in aid to coax the obstacle back toward the kidney. Or walking the patient between two assistants up and down stairs and about the room might be practised in the earlier periods of the case, with the object of facilitating the descent of the calculus into the bladder. Means of this class should be persevered in to the end, for experience is warrant that hopes may be entertained, even almost to the last, that the obstruction may be yet overcome.

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[1] Union Médicale, Nos. 69 and 70.

VI.—REACTION OF THE URINE.

There is no property of the urine of more varied and important significance than its reaction. Therewith is intimately connected the occurrence of several kinds of urinary deposits, together with the origin, growth, and medical treatment of gravel and urinary calculi.

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[1] Lately it has been recommended to open the ureter or the pelvis of the kidney above the obstruction, and so establish a permanent urinary fistula. (*See* Brit. Med. Journ., Dec. 1883.)

The reaction of the urine is liable to be affected by: *food*, the *cold bath*, *medicinal substances*, *general disease*, and *decomposition* of the secretion. It is also important not only to distinguish acid from alkaline urine, but it is at least equally so to distinguish between alkalescence from fixed alkali (potash and soda) and alkalescence from the volatile alkali (ammonia).

The most convenient method of ascertaining the reaction of the urine is by means of blue and red litmus paper. For delicate operations the violet-tinted papers are the best; and they answer both for acid and alkaline fluids—being turned red by the former and blue by the latter. To distinguish between the volatile and fixed alkali, the paper, after being rendered blue, should be allowed to dry in the open air. If the blue color persist after complete desiccation, the alkali is fixed; if it disappear, and the original color be restored, the alkalescence is due to ammonia. The smell of the urine is also a useful indication in such cases.

Healthy urine is generally acid. This arises chiefly from the presence of a number of acid salts—phosphates and urates; partly also from free acids—lactic, oxalic, acetic, etc. In a number of observations by the present writer, it was found, on an average of nineteen days, that in a healthy man it required 14.10 grains of dried carbonate of soda to saturate the total daily acidity of the urine. Some days were found throughout to exhibit a feeble acidity; on one of these only 5.9 grains of dried carbonate of soda were necessary to neutralize the whole acidity. On other days the acidity ruled high; one day the acidity equalled 22.34 grains of carbonate of soda.

The circumstances which modify the reaction of the urine may be considered under the following headings:

1. FOOD AND FASTING.—Dr. Bence Jones was the first to point out that the reaction of the urine holds a close relation to the digestion of food. He found, by examining the urine at short intervals, that a notable falling off in its acidity took place after a meal; and that in numbers of healthy persons the urine became neutral or alkaline for two or three hours after breakfast and dinner. Doubts have been thrown on the conclusions of Dr. Bence Jones by Vogel, Beneke, Sellers, and Delavaud. Some years ago I undertook a series of experiments with a view of submitting this question to a fresh examination.[1] The urine of a healthy person was examined at hourly periods after a meal, and its acidity or alkalescence carefully determined by volumetrical analysis. My results confirmed, in the fullest manner, the observations of Dr. B. Jones. A meal, whether of animal,

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[1] See a paper by the author, entitled "A Contribution to Urology," in the Memoirs of the Manchester Lit. and Phil. Soc., 1859.

vegetable, or mixed food, was found invariably to depress the acidity of the urine, and in most instances to render it actually alkaline. To this movement the name of *alkaline tide* may, for the sake of brevity, be applied. After breakfast the alkaline tide was found to set in earlier than after dinner, and its duration was more brief. In forty minutes after breakfast there appeared, nearly always, a sensible declension of acidity. The urine, however, never became actually alkaline, or even neutral, so soon. During the second hour after breakfast, the alkaline tide usually culminated; but in about a third of the observations the point of least acidity was not reached until the third hour. Then the tide turned; during the fourth hour the urine was found to be rapidly recovering its lost reaction, and toward the end of that time it had usually regained its original acidity. Thus for about four hours breakfast exercised a depressing effect on the acidity of the urine; but the secretion was not actually alkaline usually for more than an hour, sometimes for two, and very rarely for three hours.

The effect of dinner was not perceptible until the second hour after the meal. During the succeeding three hours (third, fourth, and fifth hours) the alkaline tide ran in its greatest strength. In the third and fourth hours the urine was always (with two exceptions) alkaline, when the meal had been of mixed food or animal diet. At the end of the sixth hour the tide had generally turned, and the acid reaction been restored. Three hours was the usual duration of the alkalescent state after dinner; sometimes two hours; more rarely four hours; and on one occasion five hours. The amount of free alkali discharged after dinner was generally nearly double the quantity after breakfast. This was due, probably, to the fact that dinner was usually a much heavier meal than breakfast, and its impression on the system consequently more intense.

The alkaline urine voided after food owed its reaction to fixed alkali, and not to ammonia. It was rich in earthy and alkaline phosphates. Sometimes it was clear when voided, but more commonly turbid, from the precipitation of earthy phosphates.

Although the immediate effect of a meal was thus to depress the acidity of the urine, the more *remote consequence* was to maintain and even increase the acidity. This was seen most distinctly when comparison was made of the acidity of the morning urine, when supper had been taken the night before, with that of the morning urine when no supper had been taken. In the former case the free acid discharged in the hour preceding breakfast was enough to saturate 0.88 grain of dried carbonate of soda: whereas on the mornings after supperless nights the discharge of acid was only equal to 0.51 grain.

The remote effect of animal food appeared considerably greater than that of vegetable food: so that a highly animalized diet would tend in the long run to intensify the acidity of the urine—a conclusion quite in harmony with ancient opinion.

Clinically, the urine is rarely observed to be alkaline after food. For, although it may be alkaline as it leaves the kidneys during several hours a day, after the two principal meals, it is mixed in the bladder with acid urine secreted before and after the alkaline tide, and the whole product ejected by micturition is acid. It is necessary, therefore, in order to test the effect of a meal, to analyze the secretion, as it were, by examining it at hourly intervals. It happens occasionally, however, that the urine of an ordinary micturition is the isolated product of the alkaline tide. I have known even a calculous patient, whose urine habitually deposited large quantities of uric acid, to void an alkaline urine in the forenoon from the effect of breakfast.

Dr. B. Jones considered the depression of the acidity of the urine after a meal to depend on the withdrawal of acid from the blood into the stomach for the purposes of digestion: whereby the blood became for the time less capable of yielding acid to the kidneys. On the completion of digestion the gastric juice was re-absorbed with the chyle and presently communicated its acid to the urine. An antagonism was thus supposed to exist between the stomach and kidneys; when the stomach was empty its mucous membrane was neutral, while the urine on the contrary was highly acid; but when the stomach was full, acid gastric juice was abundantly poured out on its mucous surface for the purposes of digestion, and at the same time the urine tended towards neutrality or alkalescence.

While admitting the strong probability of some such correspondence, I am disposed to attribute the occurrence of the alkaline tide after meals mainly at least to a different cause—namely, to the entrance of the newly digested food into the blood. If, as is believed, the normal alkalescence of the blood is due to the preponderance of alkaline bases in all our ordinary articles of food, a meal is *pro tanto* a dose of alkali, and must necessarily, for a time, add to the alkalescence of the blood; and as the kidneys have delegated to them the function of regulating the reaction of the blood, the urine immediately reflects any undue addition to, or subtraction from, the blood's proper alkalescence. This hypothesis is mainly supported by the coincidence of time which exists between the passage of the digested food into the blood and the occurrence of the alkaline tide. The gastric juice is poured into the stomach *immediately* after a meal, but the acidity of the urine does not suffer depression for an hour or two afterwards—not in fact until the meal has been in great part absorbed.

After the primary effect of a meal has passed off, the acidity of the urine slowly increases until food is taken again. The highest acidity is, therefore, always found after the longest fasting, or just before meals. In the early morning before breakfast, the urine was always found excessively acid, and deposited abundance of urates on cooling. There seems, however, a limit to the increase of the acidity after prolonged fasting; Dr. Bence Jones found that continuing to fast for twelve hours beyond the usual time of taking food did not intensify the acidity of the urine.

2. Effects of Medicines.—Both mineral and vegetable *acids*, when administered in large quantities, tend to raise the acidity of the urine; but their effect is inconsiderable. Urine that is habitually alkaline can certainly not be rendered acid by the internal administration of acids even in very large quantities.[1] The most powerful acidifiers of the urine are probably free carbonic acid (Heller) and benzoic acid; the latter appears in the urine as hippuric acid.

Alkaline substances have a much more powerful influence; and it is an easy matter to deprive the urine of its acid reaction and to render it strongly alkaline at pleasure. This effect may be attained by the caustic and carbonated alkalies, or by the alkaline salts of a certain group of vegetable acids—acetic, tartaric, citric, malic, and lactic acids. The most convenient for the purpose, as well as the least disturbing to the digestive organs, are the bicarbonates of potash and soda, and the acetates and citrates of the same bases. By the administration of these salts the urine may be kept persistently alkaline for weeks and months without detriment to the general health. It requires from 300 to 400 grains of the bicarbonate of potash, and about as much of the acetate and citrate, given in divided doses during the twenty-four hours, to keep the urine steadily alkaline in the adult. From numerous observations on different individuals, I found that, given in these large doses, about two-thirds of the alkali appeared in the urine as free carbonate; while the remaining third was expended in neutralizing the acidity of the urine and otherwise disposed of. The conversion of the acetates, citrates, etc., into carbonates, which was shown long since to occur by Wöhler, takes place, according to Buchheim and Magawley, in the intestinal canal, and the salts in question are therefore absorbed into the blood as carbonates. The bicarbonates, acetates, and citrates, if moderately diluted, were not found to have any tendency to cause diarrhœa; the tartrates, on the other hand, were always found to occasion more or less purging.

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[1] For further information on the action of acids on the urine, see Parkes, "On the Composition of the Urine," p. 145 *et seq.* See also a paper by Dr. Bence Jones, St. George's Hosp. Reports, 1869.

The basic phosphate of soda, the common phosphate of soda, and borax, likewise possess the power of alkalizing the urine; but their effect is very feeble, compared with that of the salts before mentioned. The common phosphate of soda, in the quantity of 640 grains in the twenty-four hours, in divided doses, produced a total alkalescence of the urine, only equal to 22 grains of carbonate of soda; whereas half the quantity of the acetate of potash produced an alkalescence equal to 120 grains of carbonate of soda; 640 grains of the basic phosphate of soda produced an alkalescence equal to 37 grains of carbonate of soda; 320 grains of borax gave an alkalescence of 9 grains of carbonate of soda; this last salt proved difficult of toleration by the stomach.

The power of alkalizing the urine is especially valuable in the treatment of urinary gravel and calculi; and to the chapter on the solvent treatment of urinary concretions I must refer the reader for further details on the subject.

3. THE COLD BATH.—Duriau[1] found that the urine became invariably alkaline after prolonged immersion of the body in a bath at a colder temperature than that of the body. Even the addition of nitric acid to the bath did not in the least alter the result; nor did the addition of carbonate of potash cause an increased alkalescence.

4. GENERAL DISEASE.—Frequent or persistent alkalescence of the urine, from fixed alkali, is an uncommon condition in any class of complaints; but a series of such cases have been recorded by Dr. Bence Jones,[2] and I have observed a considerable number myself. In persons of debilitated constitutions, in the anæmic state which sometimes follows subacute rheumatism and gout, in chlorosis, atonic dyspepsia, chronic vomiting, and even in chronic phthisis, I have seen the urine present this character. Generally, the alkalescence came and went capriciously; continuing for two or three days, and then disappearing; but presently returning again. Sometimes, however, the urine remained steadily alkaline for many weeks without intermission. In one case of this kind—a phthisical patient—the urine became acid on the occurrence of an attack of erysipelas of the head and face: it remained acid during the attack, and after its subsidence became again alkaline.

The clinical significance of alkaline urine from fixed alkali is by no means serious; it is not associated with any special morbid state, but is an occasional accompaniment of debility and spanæmia, from whatever cause arising. It is to be remembered, however, that there is a rare variety of urinary calculus composed of phosphate of lime, which must be caused by some such condition of urine as this. Individuals passing an alkaline urine are generally suitable subjects for a tonic and stimulating treatment: and, if otherwise permissible, exercise in the open air.

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[1] Archives Générales, 1856, I. 167.

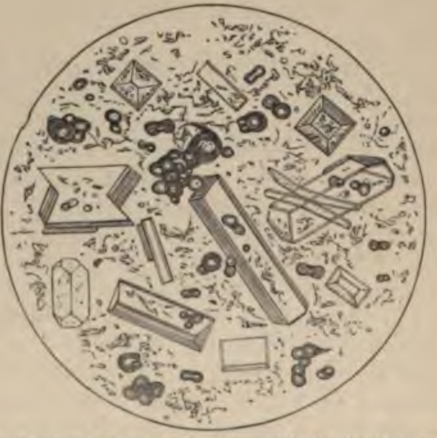
[2] Med. Chir. Trans., vol. xxxv.

5. AMMONIACAL URINE. DECOMPOSITION OF UREA—The importance of distinguishing between urine which is alkaline from fixed alkali, and that which is alkaline from ammonia, has already been insisted on. The two conditions are contrasted, not only chemically, but equally so pathologically and clinically.

Urine which is alkaline from *fixed* alkali is always secreted alkaline by the kidneys; it deposits, if at all, simple amorphous phosphate of lime, of which the particles have no tendency to accrete into gravel or calculi; it has a sweet aromatic odor; it is perfectly bland and innocuous to the mucous membranes, and is not associated with inflammation of the urinary passages.

Ammoniacal urine, on the other hand, is only in the rarest instances, and in the gravest circumstances, secreted ammoniacal by the kidneys, but usually becomes so by an after-change occurring in the lower urinary passages, or after it has been voided. Ammoniacal urine is always sedimentary; it deposits a mixture of the amorphous phosphate of lime and crystals of the ammoniaco-magnesian phosphate—sometimes, if the urine be concentrated, with the addition of lumpy spheres and rude dumb-bells of urate of ammonia (see Fig. 3). This deposit has a strong tendency to aggregate into masses or concretions; the urine has an ammoniacal and often an offensive

FIG. 3.



The normal deposit from ammoniacal urine, showing crystals of ammoniaco-magnesian phosphate, amorphous phosphate of lime, and spheres of urate of ammonia.

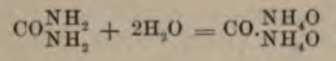
putrescent odor; it is highly irritating to the mucous membranes, and excites inflammation of them if the contact be long continued.

A urine alkaline from fixed alkali (potash or soda) reflects a state of the blood; a urine alkaline from ammonia (if alkaline when voided) points to a local affection of some part of the lower urinary passages. This latter statement is, however, not to be taken altogether without qualification. In two instances (both cases of advanced Bright's disease) I have observed the urine to be ammoniacal as it flowed from the bladder without any clinical or *post-mortem* sign of inflammation of any part of the urinary passages, or any evidence of such delay in the evacuation of the urine as might determine decomposition of it in the bladder. One of these cases is referred to hereafter (*see* Bright's Disease). Dr. Graves (*Clin. Lects.*, 1, p. 272) gives also two cases, one of continued fever, and the other of anasarca and ascites, in which the fresh urine contained large quantities of carbonate of ammonia without the least evidence of decomposition after secretion.

The transformation of urea into carbonate of ammonia is easily explained. One molecule of urea combines with two molecules of water to form one molecule of carbonate of ammonia:

$$CO(NH_2)_2 + 2H_2O = (NH_4)_2CO_3$$

or



$$CO\genfrac{}{}{0pt}{}{NH_2}{NH_2} + 2H_2O = CO.\genfrac{}{}{0pt}{}{NH_4O}{NH_4O}$$

It is now well established that the production of ammoniacal urine is an example of bacterial fermentation. Pasteur showed that urea was changed into carbonate of ammonia by the action of a minute micrococcus—to which Cohn subsequently gave the name of *Micrococcus ureæ*. The power of decomposed organic matter and of stale urine to bring about this change with celerity is simply due to the fact that these decomposing materials are highly charged with bacteria of various kinds. It is not at all probable that Pasteur's micrococcus is the only organism which acts as a urea-ferment. There are, in all likelihood, other bacteria capable of breaking up urea into carbonate of ammonia—though not, perhaps, with the same rapidity as the M. ureæ. This subject will come under notice again in the section on Microörganisms in the Urine (Chapt. iv. Sect. ix.).

If the urine be ammoniacal *when voided*, this is nearly always associated with inflammation of some part of the urinary mucous membrane—generally that of the bladder. Any condition which interferes with the complete emptying of the bladder in micturition favors the production of ammoniacal urine. Consequently, injuries to the spine determining paraplegia with paralysis of the bladder, obstinate urethral stricture, enlarged prostate, calculous concretions, morbid growths of foreign bodies in the bladder, are sooner or later complicated with ammoniacal urine. A very distressing and intractable state of things is thus brought about. The ammoniacal urine irritates the mucous membrane and induces cystitis; and the purulent secretion thus engendered reacts on the urine and favors its decomposition. The two conditions naturally aggravate each other and perpetuate each other's existence after the original cause has passed away. Cystitis may, in this way, persist for years after the removal of a stone, or the cure of a stricture, which was its original cause.

Dr. Owen Rees believes that the urine is sometimes alkaline from the secretion of an alkaline mucus by the mucous membrane of the bladder. When the membrane is irritated or inflamed, as in paraplegia from spinal injuries, extroversion of the bladder, etc., the irritated membrane pours out, as he believes, so much alkaline mucus that the reaction of the urine is changed thereby. Dr. Rees fortifies this hypothesis by an observation which he made on a case of extroverted bladder. He says: "As is usually the case in such persons, the anterior portion of the bladder was wanting; so that the fundus of that viscus covered by mucous membrane was projected forward where the abdominal walls were deficient. The openings of the ureters were thus presented to view. The mucous membrane was red and inflamed from exposure, and an alkaline fluid was constantly discharging from its surface. To what this alkaline flux amounted during the day it was, of course, impossible to ascertain; but it was more than sufficient to destroy the acidity of the urine, which was quite alkaline after flowing over the membrane. Thus a piece of blue litmus was applied to the openings of the ureters, so as to test the urine immediately it flowed from them: the paper was reddened, indicating that the urine was secreted of its natural character, and with its full amount of acidity. When, however, the litmus paper was applied about a quarter of an inch below the opening, so as to test the urine after it had passed over that short distance of mucous surface, its character was quite changed: it no longer reddened the blue litmus paper, but on the contrary was sufficiently alkaline to restore the blue color to those parts of the paper which had been previously reddened by exposure to the urine as it escaped fresh from the ureters."[1]

An opportunity occurred to me of repeating this observation on a patient with extroversion of the bladder; but I was not able to satisfy myself that the alkalinity of the exposed mucous membrane was not owing to blood-serum or lymph which oozed from the raw excoriated surface, rather than to any mucous secretion such as might be yielded by a merely inflamed mucous membrane.

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[1] Lettsomain Lectures, Med. Times and Gaz., 1851.

The therapeutical indications in cases of ammoniacal urine from decomposition within the bladder, are clear enough. The first object is to remove, if possible, the impediment to the complete emptying of the viscus. In the case of a stone or foreign body in the bladder, and in stricture, this is within reach of surgical operation. If the cause be irremovable, or if the ammoniacal urine and cystitis persist after the removal of the original cause, all our efforts must be directed to prevent the sojourn in the bladder of the stale remnants of urine after micturition; this can be best effected by completely emptying the bladder two, three, or four times daily with an elastic catheter, which the patient may be taught to introduce for himself. The bladder should also be washed out once or twice a day with an antiseptic solution—such as a saturated solution of boracic acid, or a weak dilution of carbolic acid. There is, however, another mode of washing out the bladder, which I have several times resorted to with advantage in a certain class of cases, more particularly in those in which chronic cystitis is kept up by the ammoniacal state of the urine, after the original cause has been removed. In these cases the patients are made to drink large quantities of water at regulated intervals. An abundant flow of very dilute urine is thereby kept up which effectually washes out the bladder and gradually restores the urine to its natural state.

CHAPTER III.

CHEMICAL CONSTITUENTS OF THE URINE AND THEIR VARIATIONS—INORGANIC DEPOSITS.

I.—PRELIMINARY REMARKS ON URINARY DEPOSITS AND THEIR CLASSIFICATION.

A VERY scanty, light, cloudy deposit is natural even to the healthy urine after standing some hours. This usually sinks to the bottom; but occasionally it floats like a cloud about the middle or near the surface. It is composed of epithelial scales (or remnants of them) from the mucous surfaces of the bladder and urethra, and pelvis of the kidney. Of mucus, having the usual glairy character, there is no visible trace in perfectly healthy urine.

Under a variety of unnatural circumstances more abundant deposits or sediments occur in urine; and a knowledge of their nature sometimes yields most important practical information.

Urinary deposits are arranged in two divisions—*Inorganic* and *Organic.*

Inorganic deposits include substances which, for the most part, exist naturally in the urine in a soluble state; but which, owing to their excessive quantity, or a change of reaction in the urine, or some other circumstance, are rendered insoluble, and thereby precipitated in a crystalline or amorphous condition. This division contains: Uric acid, the amorphous urates, urates of ammonia and soda, oxalate of lime, ammoniacal and earthy phosphates, carbonate of lime, cystine, leucine, and tyrosine. All these are *soluble* in mineral acids or in alkalies, and one of them (the amorphous urate) by simply warming the urine.

Organic deposits embrace all those organic forms, of which the presence alone in urine is sufficient, from their insolubility, to determine their subsidence. They do not belong in any proportion to the healthy secretion; and whenever present, they are merely suspended in it; so that when the urine is left at rest, they gravitate to the bottom and form a sediment. This group includes epithelial cells from the uriniferous tubes or from any part of the genito-urinary passages, casts or moulds (composed of a fibrinous matter) of the uriniferous tubes, oily particles, pus, blood, cancerous and tuberculous débris, spermatozoa, and bacteria. All these are *insoluble* in acids and alkalies as applied in the ordinary examination of the urine.

II.—URIC ACID, $C_5H_4N_4O_3$.

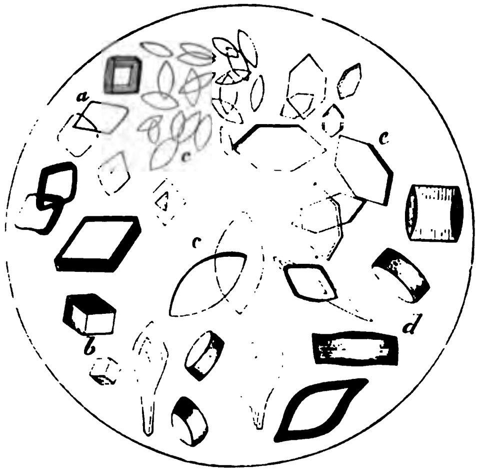
(*Synonym—Lithic acid.*)

Uric acid exists in normal urine in combination with alkaline bases; but under certain conditions it is precipitated in the free state, and forms a deposit of orange-red crystals.

Naked-eye Characters.—The crystalline nature of the deposit can nearly always be recognized by the naked eye; but in rare instances the crystals are so small that they require the microscope for their detection. Uric acid crystals may form a film on the surface, or lie scattered as brilliant brown specks on the sides of the glass, or subside into a dense red deposit like cayenne pepper. The naked eye is nearly always sufficient to identify uric acid with certainty, because no other *brown crystals* occur in urine as a spontaneous deposit. When the crystals are very minute, the deposit resembles the amorphous urate, but is denser, and sinks more rapidly. Urine depositing uric acid has commonly a rich yellow or orange color, and is invariably acid.

Micro-chemical Characters.—The primary form of uric acid is a rhombic prism or lozenge, and to some modification of

Fig. 4.

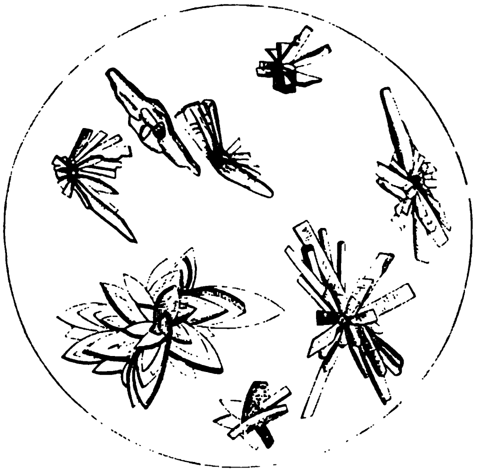


The simpler forms of uric acid crystals—quadrangular and oval tablets, cubes, six-sided tablets, lozenge and barrel-shaped figures.

this figure the protean diversities of uric acid crystals may all be referred. The angles of the crystals are sometimes almost equal, and then quadrangular tables or almost perfect cubes are obtained (Fig. 4, *a b*).

More frequently the angles are rounded off (*c d*) so as to produce ovoids and barrel shapes. A still greater elongation produces a rod, and when a number of these are joined together in a common centre, stars are produced. The beauty and endless variety of these stars are marvellous, and render them seductive microscopic objects (Figs. 5 and 6).

FIG. 5.



Stars of uric acid.

Sometimes the rays extend only in one direction, and a fan-shaped figure is produced, or two fans are joined in a common centre (Fig. 6).

Among the less common varieties may be mentioned pointed, solid-looking crystals, with a dark shading at either end (Fig. 7, *a*). When these lie flat, they have a totally different appearance, and resemble prisms of the triple phosphate (*b b*). Other forms are halbert-shaped (*c*), six-sided tablets (Fig. 4, *e*), etc. The most curious and varied forms of uric acid are generally found in albuminous urines.[1]

Uric acid is excessively insoluble. It requires 1800 parts of boiling water and 15,000 parts of cold water for solution. It is insoluble in all dilute acids, but is decomposed with effervescence by strong nitric acid. Caustic alkalies dissolve it readily, especially with the aid of heat. It dissolves also freely in weak solutions of the carbonates of lithia, potash, and soda, and in solutions of borax and common phosphate of soda. It is insoluble in alcohol and ether. It is entirely dissipated by a red

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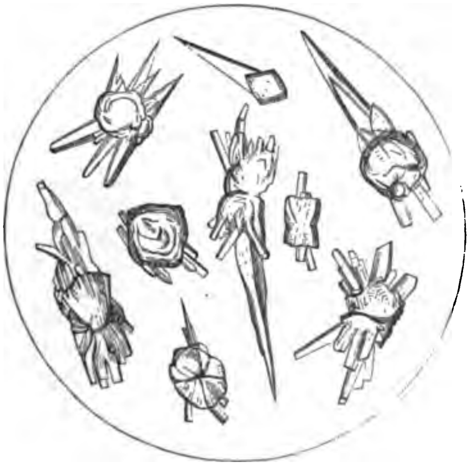
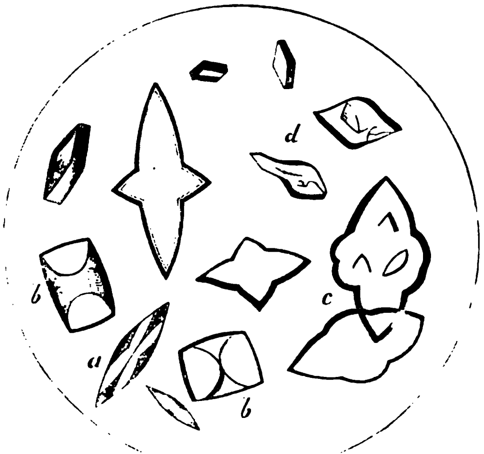
[1] Some interesting details on the varying forms of uric acid crystals and the conditions under which they appear may be found in a paper by Dr. Wm. Ord, St. Thos. Hosp. Rep., 1870, p. 335. Dr. Ord returns to this subject, and gives some additional information in a paper in the Med. Chir. Trans., 1875. See also his work, "On the Influence of Colloids upon Crystalline Form and Cohesion." Lond., 1879.

FIG. 6.



Stars and spikes of uric acid.

FIG. 7.



Rarer forms of uric acid crystals.

heat. The most delicate mode of recognizing uric acid is by the murexid test. This is performed by taking a small quantity of the suspected substance and placing it on a porcelain dish or a slip of glass; a couple of drops of strong nitric acid are then added, and the heat of the spirit-lamp applied; the uric acid dissolves with effervescence; the heat is continued until the liquid dries into a yellowish-red residue. If the residue, when cool, is touched with a rod dipped in caustic ammonia, a bright violet-blue (murexid) is instantly developed, which is perfectly characteristic.

Quantitative determinations of uric acid in urine are generally made by adding excess of acetic or muriatic acid to a known quantity of the urine, and allowing it to stand for twenty-four hours to precipitate. The acid is thrown down in a crystalline form, and may be collected either by decantation and levigation, or on a weighed filter; it is then dried and weighed. The Rev. W. Vernon Harcourt, in a long series of observations, has shown that the above process is liable to very great errors, even when the urine has been previously concentrated by evaporation. He obtained much more accurate results by the following method: Neutralize a third or fourth part of the urine of twenty-four hours, if alkaline with hydrochloric acid, or if acid with carbonate of potash; reduce this to 1½ fluidounce by evaporation; treat this with 3 drachms of hydrochloric acid combined with 1½ ounce of alcohol; decant when the uric acid has been precipitated and the liquid is clear; wash the deposit on a weighed filter first with alcohol, and when that dissolves no more, with equal parts of acetic acid and water; lastly, dry the filter and weigh.[1]

Dr. Pavy[2] estimates uric acid by making use of its reducing action on ammoniated solution of sulphate of copper (*see* Sugar). The total reducing power of the urine is first determined, and then the uric acid is precipitated by acetate of lead. The reducing power is then again determined, and the difference of the two determinations gives the reducing power of the uric acid present.

Mr. Cook finds it difficult to decide the determining point in the above method. He recommends the following process: To the alkalized urine, add a solution of sulphate of zinc, and collect the precipitate of urate of zinc which forms. Thoroughly wash the precipitate with saturated solution of urate of zinc, in order to remove urea and ammonia. The filter paper containing the precipitate may then be placed in a urea-estimation apparatus, and treated with hypobromite of sodium. The urate of zinc is decomposed, and the uric acid may be estimated by the amount of nitrogen given off.

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[1] Med. Times and Gaz., 1869, vol. ii. p. 482.

[2] Med. Chir. Trans., vol. lxiii.

(For details, *see Brit. Med. Journ.*, April, 1882.)

ORIGIN AND OCCURRENCE.—The quantity of uric acid in the urine is very minute; and were it not for its sparing solubility and liability to be precipitated both before and after emission, its clinical significance would be very slight. The daily excretion of uric acid amounts to no more than 8 or 10 grains. Individuals vary a good deal in the amounts which they habitually separate. In three healthy young students living on a similar diet and under similar circumstances, I found the following numbers:

No. 1 (mean of 47 days)	8.051 grains.
" 2 (mean of 5 days)	3.462 "
" 3 (mean of 3 days)	6.071 "

Dr. Hammond found in his own case the daily average as high as 14.14 grains.

The excretion of uric acid also presents considerable variations in the same individual from day to day. The greatest oscillation of this sort observed by myself amounted to a difference of more than one-half on two successive days: on the first day 5.45 grains were separated, and on the following day 11.7 grains. It was found that when the mode of life was tolerably uniform, the amounts separated in periods of five consecutive days varied only slightly from each other in the same individual.

The occurrence of a spontaneous deposit of uric acid is by no means a sure indication of an increased excretion; and I frequently found that those days on which a spontaneous deposit occurred, showed less uric acid than those days on which no uric acid was spontaneously deposited. The mean daily quantity of uric acid separated in twelve days on which there was a deposit was 7.7 grains; and the mean of twenty-five other days on which no uric acid was spontaneously deposited was 7.3 grains.

The digestion of food has a marked effect on the excretion of uric acid. I found it increased after eating, not only absolutely, but also relatively to the other solid matters of the urine. In the following table the results of seven days' observations on the effect of dinner are exhibited. Three periods are chosen for comparison, namely: 1, during the prevalence of the alkaline tide which corresponds with the passage of the digested food into the blood; 2, during the subsequent period in which the acid of the urine is restored, but the effect of the meal still continues to be perceptible in the considerable quantity of solid matters separated by the kidneys;[1] and, 3, during sleep, which is also a time of fasting.

Time of day. (Dinner at 2 P.M.)	Uric acid, per 1000 grains of liquid urine.	Uric acid per hour.	Uric acid, per 100 grains of solid urine.
4– 7 P.M., alkaline tide.	0.40 grain.	0.36 grain.	0.88 grain.
9–11 " acidity restored.	0.18 "	0.13 "	0.34 "
1– 7 A.M., urine of sleep.	0.39 "	0.10 "	0.60 "

It is seen that the absolute quantity hourly secreted is three times greater during the period of the alkaline tide than during the other periods; its proportion to the total solids is also very sensibly greater. Even its proportion to the water of the urine is greater than at any other period, though the urine of sleep generally (under the mode of life then followed) deposited amorphous urates very copiously after standing a few hours, whereas the urine of the alkaline tide never deposited urates.[2] It is further seen from the table that the amount of uric acid has no relation to the degree of acidity of the urine.

Professor Ranke has shown that neither sex nor age, nor the height and weight of the body, have any decided relation to the daily excretion of uric acid.[3] The season of the year, and the animal or vegetable nature of the food, have little influence, provided the articles of diet are equally rich in nitrogen. The effect of exercise is uncertain; sometimes it increases, sometimes it diminishes the uric acid.[4]

Pathologically, it is found that the daily excretion of uric acid is markedly increased in the febrile state, in certain diseases of the liver, in strumous and tubercular subjects, in rickets, scurvy, and leukæmia, and after an attack of gout. On the other hand, it is diminished during the paroxysm of gout, and, according to Ranke, after large doses of quinine.

Uric acid is nearly related, both chemically and physiologically, to urea. Uric acid yields urea as one of the products of its decomposition, both by artificial means in the laboratory and within the animal body. Nevertheless, the most exact observations have failed to show that there is any inverse correspondence between the excretion of the two substances; usually urea and uric acid increase and diminish together.

CLINICAL SIGNIFICANCE OF URIC ACID.—From what has been already stated, it will be readily conceived that the clinical interest of uric acid in the urine has not so much to do with the variations of its quantity, whether absolute or relative, as with its precipitation in the free state, and the time and place of that precipitation. The circumstances favorable to the precipitation of free uric acid are, an acid reaction of the urine, and abeyance of the conditions which determine the precipitation of uric acid in combination (amorphous urates); these latter are considered in the next section.

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[1] See Table, p. 52.

[2] The seven days' experiments here spoken of are the same seven days which are tabulated at p. 52.

[3] Ranke, Ausscheidung d. Harnsäure beim Menschen, Munich, 1858.

[4] *See* Parkes, on the Composition of the Urine, p. 88.

A deposit of uric acid occurring some twelve or twenty hours after emission has no pathological signification. Healthy acid urines usually deposit uric acid as a normal event on long standing. If the deposit take place within three or four hours after emission, the circumstance is certainly not natural; but it is not one requiring special therapeutical attention; it is frequently observed in convalescence from febrile complaints, especially articular rheumatism; also in the middle periods of chronic Bright's disease, in chorea, in certain types of diabetes, and in enlargements of the spleen.

But if uric acid be precipitated before the urine cools, or immediately after, it cannot fail to awaken apprehensions that a similar event may take place within some part of the urinary passages, and give rise to the formation of gravel and calculi, with all their train of painful and dangerous consequences. A prophylactic treatment is urgently called for under such circumstances, by which this danger may be warded off. But it will be more convenient to postpone the further consideration of this important subject to the sections which are specially devoted to the pathology and treatment of calculous disorders.

The relation of uric acid to the pathology of gout has been studied with great success by Dr. Garrod. He has proved that the blood of a gouty patient is permanently surcharged with uric acid, and that the acid is deposited in combination with soda in the cartilaginous and fibrous tissues of the joints, and becomes the cause of the articular inflammations which are characteristic of gout. This unnatural accumulation appears to be due to a defective power of eliminating uric acid in the kidneys. The kidneys themselves also suffer—their secreting tubules and the intertubular substance are clogged with urate deposits, and the foundation is laid of those atrophic changes which constitute one of the most fatal forms of chronic Bright's Disease. It may be regarded as probable that the defective power of the kidneys to eliminate uric acid in gout arises from a diminished alkalescence of the blood, and that the rational correction of this defect is, in addition to a revision of the dietary, a steady exhibition of the carbonates of lithia or potash in the intervals of the articular paroxysms.

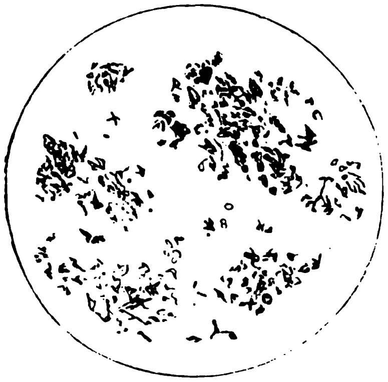
III.—AMORPHOUS URATES.

(*Synonyms—amorphous lithates; urate of ammonia of Prout and Bird; urate of soda of Heintz and Lehmann; lateritious deposit.*)

NAKED-EYE CHARACTERS.—The "amorphous urate" usually occurs as a loose, reddish, pulverulent deposit wholly devoid of crystallization. Its color is always deeper than the urine from which it falls; but the color varies extremely both in intensity and tint. It may be fawn, orange, brick-red, pink, or purplish. It commonly sinks soon and completely; more rarely, especially in albuminous urines, the precipitate continues a long while diffused in the urine, giving it a milky appearance. If the precipitation take place after the urine has been at rest in the urine glass, a film or bloom forms on the surface and sides, which is readily seen by inclining the glass to one side. By this peculiarity the amorphous urates may be distinguished from all other urinary deposits by the unaided senses.

MICRO-CHEMICAL CHARACTERS.—Under the microscope the deposit is found to be composed of minute particles or granules, coarser or finer, and more or less opaque, according to the closeness of its aggregation (see Fig. 8).

FIG. 8.



Amorphous urate deposit.

By warming the urine, the amorphous urate dissolves; the light-colored and looser deposits disappear with a slight heat, but the deeper colored and denser ones require a more elevated temperature. As no other urinary deposit disappears with simple heat, this circumstance offers an easy means of recognition. The amorphous urate answers to the murexid test for uric acid. It is decomposed by the vegetable and mineral acids (though only slowly in the cold by the former), and uric acid crystals are deposited, which may be recognized under the microscope. The urates dissolve in the caustic alkalies, and in solutions of the alkaline carbonates. They possess an intense affinity for the brown and pink pigments of the urine, which they carry down with them when precipitated; and the varied tints which they present as deposits depend on this circumstance.

The chemical composition of this deposit has been a subject of much dispute. Prout and Bird believed it to be composed of urate of ammonia, and it usually passes under that name in this country. In Germany it is commonly considered to be mainly composed of urate of soda. More recent observations indicate that neither of these views is correct; it would appear rather that the amorphous urates have not a fixed and constant composition, but vary considerably in different samples. In all, however, uric acid is combined with several bases—potash, soda, ammonia, and lime; and this is the special chemical characteristic of the deposit, that it is composed of *mixed urates.* Sometimes one base and sometimes another preponderates. The proportion of uric acid in the deposit is very large, but not constant. Scherer found a little over 80 per cent.; Dr. Bence Jones over 90 per cent. This proportion is about twice as much as is necessary to form acid urates (biurates) with the bases present; so that about one-half of the uric acid is loosely united with the biurates to form the deposit, which, therefore, resembles in its chemical constitution the quadroxalate of potash. The loosely combined uric acid can be separated from the associated biurates by simply treating the deposit with warm water, or by repeatedly washing it on a filter with cold water.

Dr. B. Jones found potash the most abundant base, next ammonia, and last soda, as the following table of his analyses shows:[1]

	First analysis.	Second analysis.
Uric acid	94.36	91.06
Potassium	3.15	3.78
Ammonium	1.36	3.36
Sodium	1.11	1.87

Hassall and Scherer always found lime in not inconsiderable quantity. Dr. Bence Jones succeeded in producing artificially exact counterparts of the amorphous urates both with potash and soda.

The precipitation of the amorphous urates depends on a conjunction of the following conditions: an acid reaction, low temperature, and concentration of the urine. The occurrence of this deposit is a sure sign of an acid reaction; and the more acid the urine, the more liable is it to deposit the amorphous urates. A drop of acetic or nitric acid will frequently determine at once the precipitation of the amorphous urates in a previously clear urine. The effect of temperature is very marked: and on cold winter mornings the urine voided on getting out of bed generally becomes turbid from precipitated urates a few hours after.

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[1] See a paper by Dr. Bence Jones in the Journal of the Chemical Society, June, 1862.

The amorphous urate deposit is not a sign of excessive secretion of uric acid by the kidneys; it indicates rather, that its proportion to the water of the urine is excessive. Urines of a high density, provided their reaction be acid and the temperature low, usually deposit urates in healthy persons. There is this difference between the conditions favorable to the deposit of free uric acid and of the amorphous urates—that a high density or (concentration) favors the latter, and a low density (or dilution) favors the former. On a comparison of the densities of a large number of urines, depositing respectively amorphous urates and free uric acid, I found the mean sp. gr. of the former 1027, and of the latter 1021. It is familiarly observed that a urine which throws down urates, will begin to deposit free uric acid a few hours after, when it has become, *quoad* uric acid, less concentrated.

The *clinical significance* of a urate deposit can be appreciated only after due consideration of the above physical and chemical conditions of its occurrence in the physiological state. I have already stated that no conclusion as to excessive elimination of uric acid can be drawn from the occurrence of the urate deposit. It has also been shown in a previous page, that during the absorption of food, and the flow of the alkaline tide, the excretion of uric acid is at its maximum, though the urine at this period very rarely deposits urates, owing to the depression or disappearance of its acidity: and conversely, that after long fasting the urine is very apt to deposit urates, because it is then concentrated and highly acid, though the hourly rate of excretion of uric acid is then at its lowest ebb.

A deposit of amorphous urates may be regarded as having either a physiological or a pathological signification. *Physiologically*, a urate deposit may be expected after profuse sweating, violent exercise, prolonged abstinence from food and drink, and in cold weather. Under these circumstances the deposit is occasional, and its color usually fawn or brownish. *Pathologically*, the most common determining cause of the precipitation of the amorphous urates is the febrile state. Even a slight degree of pyrexia, as in a common cold, is usually accompanied with a urate deposit.

The frequent or constant occurrence of a brownish or red urate deposit without, or with only a feeble degree of pyrexia, is a circumstance to awaken suspicions of some serious organic disease; but the indication is more general than special. Organic disease of the lungs, heart, liver, spleen, or any other part, attended with emaciation and waste of the tissues, is usually accompanied with abundant deep-colored urate deposit.

Functional derangements of the digestive organs are also generally accompanied by pale urate deposits in the urine. Their occurrence depends, in many cases at least, as Dr. B. Jones has indicated, on a connection between the reaction of the mucous membrane of the stomach and that of the urine.

Treatment.—From what has been stated of the determining conditions of the amorphous urate deposit, it is evident that it seldom requires direct treatment. Its indications are of more service in diagnosis and prognosis than in therapeutics. Sometimes the persistence of a urate deposit occasions such alarm to the patient that it may serve a good purpose to cause it to disappear, though no really curative end may be gained thereby. This is easily and harmlessly effected by a few two-scruple doses of citrate of potash. When this direct purpose is not aimed at, the treatment must be directed to the removal of the condition causing the deposit.

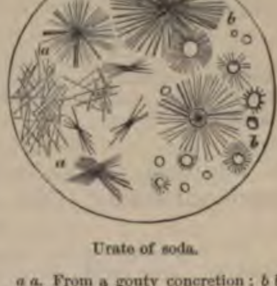
IV.—CRYSTALLINE URATES.

Urate of soda and urate of ammonia are sometimes deposited separately in urine, in the crystalline form, and under circumstances wholly different from those which determine the precipitation of the amorphous urates.

Urate of Soda.—Urate of soda is familiarly known as a constituent of gouty concretions. When the point of a lancet is thrust into one of the yellowish-white nodules so common on the ears of gouty persons, a whitish mortar-like matter escapes, which, under the microscope, is resolved into myriads of long delicate needles, arranged into bundles or stars, or lying separately (Fig. 9, *a a*).

These acicular forms are never deposited spontaneously in the urine; but they may be readily produced by adding a little liquor sodæ to the common amorphous urate, in a watch-glass, and allowing the solution so formed to concentrate by evaporation in the air (Fig. 9, *b b*).

Fig. 9.



Urate of soda.

a a. From a gouty concretion; *b b*. Artificially prepared by adding liq. sodæ to the amorphous urate deposit.

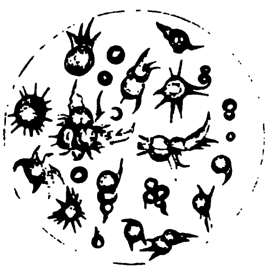
Urate of soda is a comparatively rare spontaneous deposit in urine. It occurs, however, occasionally in gout, and in the febrile state, especially in children. It forms a whitish or yellow sediment, which sinks rapidly; it is associated with an acid reaction of the urine, and is frequently, if not generally, deposited in the bladder before the emission of the urine. In this respect it differs from the amorphous urate, which is never deposited until the urine has cooled.

Under the microscope the spontaneous deposit of urate of soda exhibits irregular, opaque, globular, and lumpy masses, from which project spiny crystals, sometimes straight, sometimes variously curved (*see* Fig. 10).

The occurrence of this deposit in the febrile complaints of infants and children probably depends on the urine being excessively scanty and concentrated and long detained in the bladder. Its appearance in such cases is temporary, and ceases on the reëstablishment of the flow of urine. The annexed drawing (Fig. 10) was made from a deposit voided by a little child of three years. The child was suffering from severe infantile remittent, and no urine had been passed for two days. While I was examining the abdomen, the child cried, and the urine began to flow. The first portions were turbid and of a gamboge-yellow color, and contained the spiny masses here delineated; after about an ounce of this had come away, several ounces of clear high-colored acid urine followed.

Clinically, this deposit derives its chief importance from the circumstance that it is precipitated within the urinary passages. The spiny crystals irritate the mucous membrane of the bladder or urethra; and the latter canal may even be blocked up by impaction of masses of the deposit. It may also form a nucleus around which calculous matter may hereafter aggregate. The great comparative frequency of vesical calculi in children is not improbably owing to the occurrence of this deposit in the numerous fugitive febrile attacks to which children are subject.[1]

FIG. 10.



Hedgehog crystals of urate of soda, spontaneously deposited from the urine of a child.

URATE OF AMMONIA.—When urine becomes strongly ammoniacal, it is liable to precipitate urate of ammonia, in addition to the mixed phosphates which are necessarily deposited under those circumstances. The urate of ammonia has usually a dense white color; but I have known it possess a beautiful violet hue.

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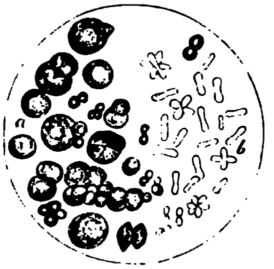
[1] The correctness of this conjecture is fully borne out by the researches of Dr. Vandyke Carter on the Structure and Formation of Urinary Calculi. He found that urates formed the chief part of the nucleus in the majority of urinary calculi. (On the Microscopic Structure of Urinary Calculi, Lond., 1873.)

Two forms are seen under the microscope. The most common are spheres and globular masses, which appear almost black by transmitted light, owing to their opacity (*see* Fig. 11, *a*). These spheres are easily obtained by leaving a urine containing the amorphous urate to stand in the air until it becomes ammoniacal. The second form (*b*) occurs as very minute slender dumb-bells: these generally lie singly; or two lie athwart each other so as to form a cross; or three are united so as to form a rosette. They become coarser and larger with long keeping of the urine. This deposit has no special *clinical significance;* its occurrence is merely an incident in the ammoniacal decomposition of the urine. It is a frequent ingredient of the secondary phosphatic crust which invests urinary calculi in the later periods of their growth (see Urolithiasis).

FIG. 11.



Urate of ammonia spontaneously deposited. *a.* Spheres and globular masses; *b.* Dumb-bells, crosses, rosettes.

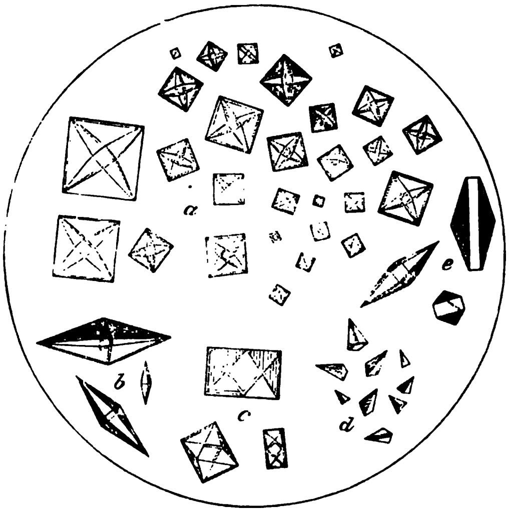
V.—OXALATE OF LIME.

(*Oxaluria; oxalic acid diathesis.*)

NAKED-EYE CHARACTERS.—A deposit of oxalate of lime is usually very scanty, and looks like a slight cloud of mucus. Owing to this, and its colorlessness, it seldom attracts the attention of a patient. If, however, the urine be transferred into a urine-glass immediately after emission, as is usually practised in hospital wards, the following appearances are produced, which are sufficiently characteristic to enable the observer to recognize the deposit with certainty by the unaided eye. The sides of the glass are seen to be traversed by very numerous fine lines, running in bands, transversely or obliquely, giving an appearance as if the glass were finely scratched. This appearance is due to the crystallization of the oxalate on the fine lines or inequalities left after cleaning the glass by towelling. The subsided portion is equally peculiar; it consists of two parts—a soft, pale gray, mucous-looking sediment, occupying the bottom of the vessel, and overlying this a snow-white denser layer with an undulating but sharply limited surface. The only other substance which crystallizes in lines on the sides of the glass is uric acid; this is easily discriminated by the greater coarseness of the lines and their more or less brown color.

Micro-chemical Characters.—Oxalate of lime occurs in very minute crystals, the largest only appearing to the naked eye as sparkling points. Two forms are met with. The most common are octahedra, greatly shortened, or flattened, in one direction. The crystals present different appearances according to the side on which they lie. Commonly they rest on their short axis, and appear as squares crossed diagonally by a pair of lines (Fig. 12, *a*). As they roll over in the field of the microscope, they assume various forms—lengthened, pointed octahedra, crossed parallelograms, etc. (*b*, *c*). Sometimes half-crystals are seen—four-sided pyramids on a square base (*d*); and sometimes two such pyramids, instead of being united by their bases to form the ordinary octahedron, are separated by a short square prism (*e*). The second form of oxalate of lime is that of dumb-bells and minute ovoids and circles (Fig. 13). The different appearances are produced by the different postures assumed by the objects; and, as they roll over in the field of the microscope, the dumb-bell is seen to change to an ovoid or circle, and *vice versâ*. Their real shape is that of an oval or circular disk, with rounded margins, and a depression in the centre on either face.

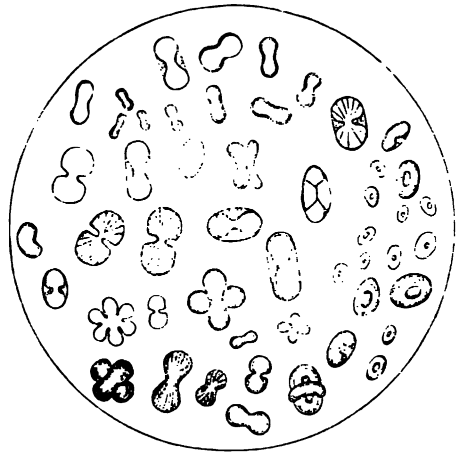
Fig. 12.



Oxalate of lime. *a*, *b*, *c*. Octahedra in various positions; *d*. Pyramids; *e*. Pyramids with intervening square bases.

The dumb-bells are probably identical in composition with the octahedra. Dr. Bird, in his later editions, expressed a doubt on this point, on the ground of their different behavior with polarized light,[1] and suggested that they consisted of oxalurate of lime. Schunck has recently shown that oxalurate of ammonia may be found in normal urine.[2] If this prove to be universally true, it would afford an easy explanation of the frequent occurrence of oxalate of lime in urine. The precipitation of oxalate of lime as dumb-bells depends on some physical condition which interferes with the ordinary crystallization. Very frequently urine depositing dumb-bells contains little masses of viscid mucus; and it seems probable that a certain viscidity of the urine is essential to this globular precipitation.[3]

FIG. 13.



Dumb-bells and ovoids of oxalate of lime.

Oxalate of lime is insoluble in alcohol, ether, water, and the vegetable acids; but it dissolves readily in the mineral acids. The urine depositing it is usually high-colored and acid; very rarely neutral or faintly alkaline; and never, so far as I have seen, freely alkaline. Oxalate of lime is often conjoined with uric acid and the amorphous urates; much more rarely with the stellar phosphate of lime.

PRODUCTION AND OCCURRENCE.—The frequent occurrence of oxalic acid in the urine cannot be a matter of surprise when it is remembered that it differs from carbonic acid—one of the chief final products of the disintegration of the tissues—only in possessing half an atom less of oxygen. It constitutes probably one of the penultimate stages in the series of decompositions through which the effete tissues pass preparatory to their final exit from the body. A large number of substances which occur in the body (uric acid, creatin, fats, starch, sugar, etc.) can be made to yield oxalic acid in the laboratory; and it is highly probable that a similar change occurs in the living economy. With regard to uric acid, this has been positively ascertained by Wöhler; and Dr. Garrod has succeeded in showing that oxalic acid is present, sometimes at least, in the blood.

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[1] Thudichum states that octahedra of oxalate of lime do polarize light, and that there is no reason to believe that the dumb-bells differ from them in composition.

[2] Proceedings of Roy. Soc., 1867.

[3] The precipitation of carbonate of lime in spheres and close dumb-bells in the viscid urine of the horse is an example of the same kind. Mr. Rainey has shown a much wider application of the same principle in the calcifications which take place naturally in the hard tissues of the body. *See* Med.-Chir. Rev., vol. xx. p. 451.

It is therefore easy to understand how oxalic acid should exist in urine; also that it may be partly derived from the blood and appear in the urine at the moment of secretion, and partly be produced after the urine is secreted by conversion from uric acid. Dr. Owen Rees has nevertheless expressed his strong disbelief in the existence of oxalate of lime in the blood, apparently on the ground of the chemical difficulty in conceiving that oxalate of lime, from its insolubility, could exist dissolved in the blood; he contends that all the oxalate of lime found in urine is produced from uric acid after separation from the blood.[1] These theoretical objections, however, do not avail against the positive fact, that oxalic acid and its compounds, even the insoluble oxalate of lime, pass through the blood into the urine when introduced into the stomach. Wöhler found that oxalic acid given to dogs caused oxalate of lime to appear in the urine. Piotrowsky confirmed these results by experiments on himself. He took, in divided doses, from 80 to 100 grains of oxalic acid in the course of about six hours, and found that from 8 to 14 per cent. appeared in the urine as oxalate of lime, mixed with a little alkaline oxalate. Similar results were obtained with the oxalate of soda. When the insoluble oxalate of lime was taken in the same doses, very much less of it appeared in the urine; still about 1½ per cent. could be recovered.[2]

CLINICAL SIGNIFICANCE.—Distinction must be made between slight occasional deposits, and large quantities occurring persistently. In the former case, it cannot be said positively that there is any departure from the normal state, seeing that oxalic acid is in all probability a natural constituent of urine; at least, it is constantly found in the urine of perfectly healthy individuals.

But when the deposit is *constant and large*, an abnormal state must be recognized to exist; and we are called upon to consider what pathological significance it may have, and whether it supplies any indications for treatment.

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[1] "On Calculous Disease." Croonian Lectures for 1856, pp. 2 et seq.

[2] Archiv f. Physiol. Heilk., 1857, p. 122. Dr. Leared and Dr. Dyce Duckworth have also found that taking ℥iij of lime-water or a grain of oxalic acid caused oxalate of lime crystals to appear in the urine of healthy persons. St. Barthol. Hosp. Rep., 1866, p. 160; and Med. Times and Gaz., 1867, I. 219.

The most obvious inference is, that there exists in such a case a liability to the formation of an oxalate of lime calculus. This point, and the preventive treatment to be followed, will be considered in the section on calculous disease.

But a much wider significance has been given by some authors to oxalate of lime deposits; and a certain group of symptoms which are alleged to accompany these deposits, has been erected into a distinct pathological state under the name of the *oxalic acid diathesis*. Dr. Prout was the first to promulgate this view; and he has been followed by Dr. Bird and Dr. Begbie. Dr. Bird gives the following account of the symptoms which accompany oxaluria: "They" (the patients) "are generally much emaciated, excepting in slight cases, extremely nervous, painfully susceptible to external impressions, often hypochondriacal to an extreme degree, and in very many cases labor under the impression that they are about to fall victims to consumption. They complain bitterly of incapability of exerting themselves, the slightest exertion bringing on fatigue. Some feverish excitement, with the palms of the hands and soles of the feet dry and parched, especially in the evening, is often present in severe cases. In temper they are irritable and excitable; in men the sexual power is generally deficient and often absent. A severe and constant pain, or sense of weight across the loins, is generally a prominent symptom, with, often, some amount of irritability of the bladder. The mental faculties are generally but slightly affected, loss of memory being sometimes more or less present." ("Urinary Deposits," 5th ed., p. 251.)

This train of symptoms is familiar enough to every practitioner: and the occurrence of oxaluria in such cases is undoubtedly common enough ; but these symptoms may be present in typical completeness without oxaluria, and conversely oxaluria may exist in its highest intensity, and even go on to the formation of a mulberry calculus, without evoking any of the abovementioned symptoms. Every one who has had experience in calculous disorders cannot have failed to observe that the subjects of mulberry calculus, especially children, are not unfrequently in the enjoyment of blooming health so long as no local irritation has been set up by the concretion. It will also not fail to be remarked that the symptoms attributed to oxaluria are almost identical with those attributed to spermatorrhœa. Disturbed equilibrium and loss of tone of the nervous system, with symptoms (more or less intense) of impaired digestion, are unfortunately a too common resultant of the intense activity of mind and body, and the trying wear and tear of modern life: and both physician and patient are naturally anxious to find some material alteration to account for a condition which is sufficiently serious, and which is remarkable for its want of definition. The patient often fixes on some derangement of the sexual function, generally, in these times, on spermatorrhœa, under the inspiration of unscrupulous publications too widely circulated among the curious public; or on heart disease, consumption, or gravel. The physician is able by means of physical examination to set aside these more open delusions, but falls himself into the trap of his own ingenuity, and is only more elaborately wrong than his patient. He finds crystals of oxalate of lime in the urine, and persuades himself that he has discovered the first link in the chain of consequences. It may be much questioned (and I certainly see no necessity for such a supposition) whether there be any morbid condition antecedent to the plain symptoms of the case, namely, an overtasked and disturbed nervous system, and a mismanaged and deranged digestion.

The facts and considerations which lead to the above reflections are:

1. Intense oxaluria may exist persistently without evoking the group of symptoms attributed to the oxalic diathesis.

2. This group of symptoms may exist in typical development without the occurrence of deposits of oxalate of lime in the urine.

3. The most varied morbid states are found to coexist with oxaluria. I have been in the habit for many years of noting the symptoms and pathological states of those patients in the Manchester Infirmary who had pronounced oxalate of lime deposits. Five out of every six exhibited none of the symptoms attributed to oxaluria. Almost every variety of disease was occasionally found associated therewith. The following especially were observed: chronic phthisis, cardiac affections, emphysema with chronic bronchitis, chronic rheumatism, anæmia, hemiplegia, malignant disease of the liver and stomach, chronic vomiting, and cirrhosis.

I am strongly convinced, that oxaluria arises from a variety of conditions—many of them not accompanied by appreciable departures from health—in which the assimilation of food or the disintegration of the tissues goes on imperfectly; and that it is impossible to assign any constant train of symptoms as the cause or the consequence of oxaluria. At the most, oxaluria is only one in a long list of symptoms, and one of the least significant.

Beneke, who has subjected this question to an elaborate examination, both in the way of experiment and observation, has formulated the following propositions, which appear to me to be well founded:

1. Oxaluria, a condition which accompanies the lighter or severer forms of illness, has its proximate cause in an impeded metamorphosis—that is, in an insufficient activity of that stage of oxidation which changes oxalic acid into carbonic acid.

2. Oxalic acid has, if not its sole, its chief source in the azotized constituents of the blood and food; everything, therefore, which retards the metamorphosis of these constituents occasions oxaluria.

3. Such a retardation of the metamorphosis of the azotized constituents of the blood may be determined by the following causes:

a. Abuse of azotized articles of food (direct retardation).

b. Abuse of saccharine and starchy articles of food (indirect retardation).

c. Insufficiency of the red blood-corpuscles and (eventually) diminished oxidation.

d. Insufficient enjoyment of pure, fresh, ventilated air.

e. Organic lesions which in any way impede respiration and the circulation of the blood.

f. Conditions of the nervous system which bear a character of depression, whether these arise primarily from mental derangement or from pathological states of the blood.

4. Excess of alkaline bases in the blood, which, as numerous observations tend to show, plays an important part among the etiological conditions of oxaluria; and it is not improbable that an increased production of lactic and butyric acids in the digestive canal, consequent thereupon, impedes the development of the red blood-corpuscles, and thereby generates that chlorotic state which so often occasions and accompanies oxaluria.

5. Catarrhal conditions of the intestinal mucous membrane, in case they are accompanied by oxaluria, have at most only a common source. They may determine oxaluria by causing deranged digestion, but cannot be considered as its proximate cause.[1]

TREATMENT.—After the foregoing reasoning and conclusions, it is scarcely necessary to say that oxaluria does not, in the opinion of the present writer, furnish special indications for treatment; nevertheless it will be found that, apart from the existence of organic disease, the conditions most frequently found associated with oxaluria, varied as they are, call for a tolerably uniform therapeutical action. They demand a quickening of the oxidation processes, and a careful regulation of the diet. The skin should be encouraged to activity by systematic use of cold sponging, friction of the skin with flesh-brushes, wearing of flannel vests and drawers, regulated exercise in the open air —if available, horse exercise. Many of the cases yield only to repeated change of air; the bracing atmosphere of upland and sea-side localities generally suits the best. It will often be found advantageous to withdraw for a time the use of tea and coffee, and to substitute milk; or if this prove heavy, milk mixed with one-fourth of lime-water. The diet should be judiciously compounded of due proportions of animal and vegetable substances —diminishing the one or the other group of aliments according to the ascertained idiosyncrasy of the patient. He must be cautioned against heavy meals, and trained to partake more moderately of four meals a day. Digestion may be promoted by the administration of the mineral acids in light bitter infusions, or by small doses of the bicarbonate of potash in the same combination. It is not easy to determine beforehand which of these opposite medicaments will prove most grateful to the stomach. The rule of choice is, to administer the acid when the dyspeptic symptoms point to an atonic state of the organ and of the body generally, and the alkali when the signs point to gastric and general irritation.

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[1] Zur Entwicklungsgeschichte d. Oxalurie, by F. W Beneke. Göttingen, 1852.

VI.—CYSTINE ($C_3H_7NSO_2$).

(*Synonym—cystic oxide.*)

Cystine or cystic oxide is a crystalline body of great rarity, which is found only under certain abnormal conditions in the bodies of animals. Hitherto it has been detected with certainty only in man and the dog. Cloetta asserts that he found it once in the kidneys of an ox.

Cystine was discovered by Wollaston in 1805, in a urinary calculus which was mainly composed of it. Since that time a considerable number of cystine calculi have been found in different parts of Europe and America; but, as compared with other urinary concretions, this is one of the most rare.

As a urinary deposit, cystine has been even less frequently met with than as a calculus; and as nothing is known touching the organic processes and constitutional states in which cystine is produced, the clinical interest attaching to it is for the most part confined to its manifestations as gravel and calculus. A number of cases have, however, been observed where cystine existed simply as a urinary deposit, or dissolved in the urine.[1]

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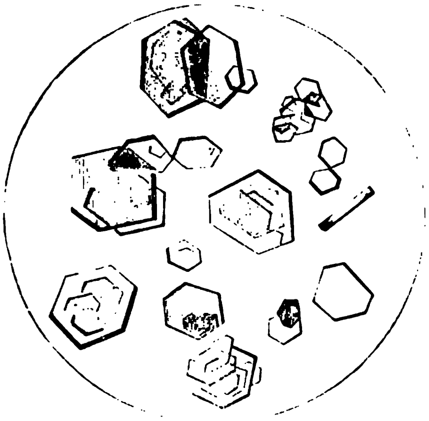
[1] Niemann (Deutsch. Arch. f. klin. Medicin, Bd. xviii. p. 232) has collected 52 cases of cystinuria. The following additional cases may be referred to: Loebisch, Liebig's Annalen, 182; Southam, Brit. Med. Journ., II., 1876, and II., 1878; Guyot, Progrès Médical, 1878, No 10; Ebstein, Deutsches Arch. f. klin. Med., Bd. xxiii.; Ultzmann, Med. Pr., No. 29, 1878; Wood, Bost. Med. and Surg. Journal, 1878.

The following case of cystine calculus with concurrent cystinuria occurred in the Manchester Infirmary:

J. M., æt. 57, admitted June 15, 1874. There was no history of stone or gravel in any member of his family. He was quite healthy until January, 1873, when he suffered from repeated attacks of renal colic. In April, 1873, symptoms of vesical calculus appeared, and these have continued ever since. On sounding, a stone was found in the bladder. The urine contained a good deal of pus, and under the microscope numerous well-formed hexagonal crystals of cystine were detected (Fig. 14).

FIG. 14.



Crystals of cystine spontaneously voided with the urine of J. M.

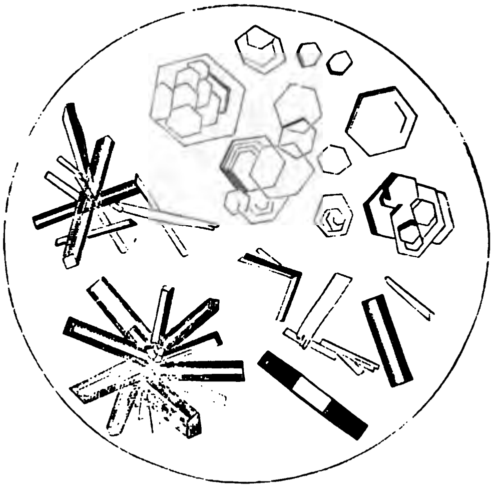
This man was subjected to one sitting with the lithotrite by Mr. Southam. Much irritation of the bladder followed, with severe bronchitis, of which the patient died a fortnight after the operation. After death extensive pyelitis with sacculation of the kidneys was found; and broken portions of a cystine calculus were discovered in the bladder.

Generally, urine depositing cystine is turbid when voided; and on standing, a copious light sediment subsides, much resembling (to the naked eye) fawn-colored lithates. The urine from which cystine is deposited has sometimes a peculiar sweetbriar odor, a honey-yellow color, and an oily appearance. It is usually faintly acid and very liable to spontaneous decomposition, in the course of which it evolves sulphuretted hydrogen, and blackens white glass vessels. Dr. Golding Bird observed that urine containing cystine changed from yellow to green when it became decomposed.

A few drops of acetic acid always precipitate an additional quantity of cystine from the supernatant urine; and if a urine containing cystine holds it all in solution, as may happen when the quantity is very small, acetic acid throws it down.

A deposit of cystine is not dissolved by heat, nor by the vegetable acids. It is instantly dissolved by caustic ammonia, and if the solution be exposed in a watch-glass to evaporation in the air, beautiful six-sided crystals are obtained as the volatile alkali exhales (Fig. 15). This is the characteristic reaction of cystine, and leads to its easy identification. Cystine is also soluble in the carbonates of the fixed alkalies; but not in carbonate of ammonia, which, indeed, is its best precipitant from acid solutions. It is soluble also in the mineral acids, but insoluble in acetic and tartaric acids. It is insoluble in water and alcohol. Heated on platina foil, it evolves thick white fumes, having a peculiar offensive odor resembling garlic.

FIG. 15.



Cystine. Hexagonal tablets and prisms from an evaporated ammoniacal solution.

Cystine is a body of very weak affinities, without taste or smell; it acts as a feeble base, and forms crystalline compounds with nitric and hydrochloric acids. According to Pelouze, it may also play the part of an acid; he obtained two compounds with silver, which he denominated cystates.[1]

A spontaneous deposit of cystine in urine is composed of hexagonal tablets. When the ammoniacal solution of cystine is allowed to evaporate in the air, magnificent crystals are obtained, which furnish brilliant objects for the microscope. Cystine is dimorphous, and crystallizes in two forms, namely, as six-sided tablets and square prisms (*see* Fig. 15).

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[1] "Note sur la cystine," by Pelouze, appended to Civiale's Mémoire sur les calculs de cystine, at p. 441 of Civiale's treatise Du traitement médical de la pierre. Some further consideration respecting the constitution and physiological relations of cystine may be found in a paper, by J. Dewar and A. Gamgee, in the Journ. for Anat. and Physiol., 1870.

The ammoniacal solution generally deposits hexagonal plates only, or these mixed with a few prisms; sometimes, however, the prisms are more abundant than the plates. The prisms either lie singly or form stars: they refract light strongly, and the facets which lie slantingly out of the direct line of vision appear perfectly black, contrasting with the brilliant lustrous white of the planes through which the light passes vertically. This gives a peculiar striped appearance to the prisms, and causes them to appear deceptively six-sided. The hexagonal tablets have an iridescent mother-of-pearl lustre; their surfaces are often beautifully chased by lines of secondary crystallization; they also form thick rosettes of great brilliancy.

The production of cystine in the animal body has as yet received no elucidation. It may, however, be assumed that it preëxists in the blood, and is merely separated by the kidneys. The most remarkable fact respecting the constitution of cystine is the large amount of sulphur (nearly 26 per cent.) which it contains. The close analogy of composition between it and taurine, renders it not improbable that the liver is the original source of cystine;[1] the discovery of cystine in the livers of typhus patients by Scherer[2] lends support to this view; and Marowsky[3] also found cystinuria accompanying a diminished secretion of bile. Later researches, however,[4] have thrown doubt on this theory.

Ebstein has reported a case in which cystinuria, accompanied by albuminuria, suddenly occurred in the course of joint rheumatism, the joint pains at the same time diminishing. After about a fortnight's duration, the albumen and the cystine suddenly disappeared from the urine.

The other constituents of the urine have not been found altered in any constant manner in cystinuria; and the later

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[1] The close connection between cystine and taurine may be gathered at a glance by a comparison of their composition per cent.:

	Cystine.	Taurine.
Carbon	30.00	19.20
Hydrogen	5.00	5.00
Nitrogen	11.66	11.20
Oxygen	26.66	38.40
Sulphur	26.66	25.60

[2] Archiv f. Path. Anat., Bd. x. p. 228.

[3] Deutsch. Arch. f. klin. Medicin, Bd. iv. p. 449.

[4] *See* Niemann, loc. cit. p. 232.

analyses of Beale[1] and Toel[2] tend to support the original opinion of Civiale, that in cystinuria, as in most other calculous states, the composition of the urine, apart from the dominant calculus-forming constituent, is normal. It would be of interest to determine the amount of unoxidized sulphur voided with the urine in these cases. When it is remembered that from 3 to 5 grains of unoxidized sulphur are daily discharged with the urine by healthy men,[3] it would seem *à priori* not improbable that cystine is only the sulphur extractive in a new form. If it be so, one would expect the unoxidized sulphur to be diminished in cystinuria. The excretion of sulphuric acid was not found diminished in a case examined by Beale. Loebisch and Niemann, however, have observed in their cases, that the total amount of sulphuric acid excreted, calculated absolutely and also in relation to the nitrogen present, was somewhat less than normal.

Niemann also found a diminution of the uric acid excreted, but Ebstein came to the conclusion that a diminution of urea, uric acid, or sulphuric acid could not be proved in cystinuria.

Deposits of cystine are very bulky: but the quantity, when weighed, is found unexpectedly small. Percentage determinations have been made by Prout and Beale. The former found 0.024, and the latter 0.09. Loebisch and Niemann have each determined the daily excretion, and have obtained as the daily average 0.393 grain and 0.509 grain respectively.

Cystine may persist in the urine for many years; it may disappear for a while, and reappear again after a longer or shorter interval; or it may disappear permanently. It is sometimes succeeded by deposits of uric acid. The connection of cystine in the urine with deposits of the earthy phosphates, on which Prout and Civiale insist, is probably nothing more than a coincidence depending on the strong tendency of urine containing cystine to decompose and become ammoniacal, whereby the phosphates are necessarily precipitated.

One of the most curious circumstances in the history of cystine is the unquestionable tendency which it shows to run in families. The facts bearing on this point will be referred to in treating of cystine calculus.

Cystine has been found more commonly in males than in females, and mostly in children and young adults; though no age is exempt. Dr. Shearman, of Rotherham, believes that scrofulous children and chlorotic females are especially liable to cystinuria. In a young woman from whom Mr. Jordan extracted a cystine calculus some years ago, in the Manchester Infirmary, I found considerable tuberculous consolidation of both apices. The more recent researches of Fabre[1] do not support the opinions of Dr. Shearman. Fabre examined the urine of a large number of tuberculous persons and of thirty-six strumous children, but failed to detect a trace of cystine. In fifteen chlorotic females he likewise obtained negative results.

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[1] Urine and Urinary Calculi, p. 311.

[2] Ann. der Chem. u. Pharm., Bd. xcvi. p. 24.

[3] Ronalds: Phil. Trans., 1847, p. 461. The observations of Ronalds have been since confirmed by Griffith and Parkes. The same has been found in the urine of dogs by Bischoff and Voit.

It is undoubted that persons may void cystine for years, without any other deviation from health than what is caused by the physical irritation of the concretions, when these form. The brothers Planta, operated on by Civiale for immense cystine calculi, were known to have been excreting cystine in quantity for six years continuously, without any impairment of health. The sisters observed by Toel looked well, and were perfectly healthy, except that they were liable to nephritic pains from time to time, when they passed small calculi and gravel.

The *clinical significance* of cystine is therefore chiefly, if not wholly, the danger of the formation of stone and gravel.

The *treatment* of cystinuria, apart from that which is designed to prevent the formation of concretions, is necessarily, so long as the *rationale* of its production is so obscure, unsatisfactory. Dr. Prout believed he saw benefit from the long-continued use of nitro-muriatic acid. Dr. Bird, on the other hand, found the same remedy useless. If chlorosis or struma coexist with cystinuria, these will of course demand their appropriate treatment; but as yet nothing is known which can pretend to have any direct influence in checking the formation of cystine.

VII.—XANTHINE ($C_5H_4N_4O_2$).

(*Synonyms—xanthic oxide; uric oxide.*)

This rare substance was originally discovered by Dr. Marcet, about the year 1817, in a urinary calculus given to him by Dr. Babington. This concretion weighed only 8 grains, and had apparently been passed spontaneously. In 1816 the elder Langenbeck removed from a peasant boy, eight years of age, a stone as large as a small egg, which was afterwards identified by Stromeyer with the xanthic oxide or xanthine of Marcet. In 1837 a portion of this stone was analyzed by Liebig and Wöhler;[2] in 1846 it was reëxamined by Bodo Unger[3] with identical results. The name Xanthine was originally used by Unger to designate a substance found by him in guano, which he at first considered identical with Marcet's xanthic oxide, but which he subsequently established as a new substance under the name of guanine; the name xanthine then passed permanently to Marcet's xanthic oxide.

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[1] A. Fabre: De la cystine, etc. Paris Thesis, 1859. Fabre calls attention to the hexagonal appearance of uric acid crystals when precipitated by acetic acid; and he attributes the conclusions of Shearman to confounding these with cystine crystals.

[2] Poggend. Ann. der Physix, 1837, Bd. xli. p. 393.

[3] Liebig's Ann. der Chem. und Pharm., Bd. lviii. p. 17.

In 1829, Laugier[1] described some minute calculi obtained from a patient who had passed several. Three of these were handed over to Laugier; the largest of them weighed less than one-sixth of a grain. Their deep yellow color, their spherical form, their smooth surface, seemed to indicate that they consisted of uric acid. They proved, however, to be xanthine, and yielded the characteristic reaction with nitric acid and potash.

Professor Dulk, of Königsberg, removed a xanthine calculus weighing 7 grains from the urethra of a boy (Bird).

In 1866, Mr. T. Taylor discovered in the Museum of the London College of Surgeons a calculus composed of nearly pure xanthine. When entire it weighed 90 grains. It was extracted from a Mussulman child, four years old, by Mr. Coles, a surgeon in the employ of the East India Company.[2]

Xanthine is a substance closely connected with uric acid, differing from it in composition only in possessing one atom less of oxygen. Xanthine has been discovered by Scherer in the blood; also in the muscles, liver, spleen, and brain. Scherer[3] further states that a very minute quantity of xanthine is a natural constituent of healthy urine. Heller has been unable to convince himself of the correctness of this statement.[4]

Xanthine has been met with five times (as above recorded)[5] as a urinary calculus: as a urinary deposit it is alleged to have been encountered by Bird, Douglas Maclagan, and Bence Jones. Maclagan found it mixed with earthy phosphates in the urine of a hysterical girl.[6] Dr. Bence Jones's case was a school-boy between nine and ten years of age. Three years before he had suffered an attack resembling nephritic colic, but without subsequent passage of a stone. When first seen, the urine made at night contained a small quantity of albumen, but that of the morning contained none. A month later the urine was found "quite thick and deep colored. A drop was placed under the microscope, and a crystalline deposit was found resembling one form of uric acid. From this form I considered the deposit was uric acid—(the crystals were pointed ovals). On examining the unfiltered urine for albumen by heat, I was surprised to see the crystalline deposit entirely dissolve. A fresh portion of sediment showed the same crystalline appearance and the same solubility by heat. . . . A day or two afterwards another specimen was brought to me, containing the same crystalline deposit soluble by heat. The sediment formed about an eighth of the bulk of the fluid. It was collected on a filter, washed with alcohol, and it gave the following reactions: It dissolved in water and hydrochloric acid; when treated with nitric acid it dissolved without effervescence, and when evaporated to dryness it left a yellow residue."[1] Further examination of the urine on subsequent occasions yielded no traces of xanthine.

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[1] Journ. de Chim. Méd., vol. v. 1st series.

[2] Path. Soc. Trans., xix. 275.

[3] Liebig's Ann. d. Ch. u. Ph., Bd. cvii. Heft 3, 1858.

[4] Heller's Harnconcretionen, p. 131, note.

[5] A short mention of two other cases which occurred in America is found in the Brit. Med. Journ , Jany. 1883, p. 148.

[6] Edin. Med. Journ., 1858, p. 121. Scherer doubts this case; and thinks the reaction mentioned by Maclagan insufficiently characteristic.

Jackson thought he detected xanthine in diabetic urine; but the tests he relied on were untrustworthy. Lehmann was unable to detect xanthine in several diabetic urines which he examined.

Purified xanthine, according to Städeler (who operated on xanthine obtained from Langenbeck's calculus), shows itself under the microscope as very small irregular granules. When dried it forms brittle crusts, almost chalk-white, with a slight tinge of yellow, which become deeper-colored when powdered. When rubbed, xanthine acquires a waxy lustre. It is soluble in alkalies, also moderately freely in concentrated and warm hydrochloric acid. This solution becomes turbid on cooling, and deposits quadratic octahedra of a combination of xanthine with the acid. It dissolves without effervescence in nitric acid, and the solution on evaporation leaves a bright yellow residue, which becomes violet-red when treated with solution of caustic potash.[2] The solubility of xanthine in water is subject to extraordinary variations, which are not yet understood. Städeler found pure xanthine from Langenbeck's calculus to dissolve in 13,333 parts of cold and in 1178 parts of hot water. Strecker found artificial xanthine, prepared by him from guanine, to vary in its solubility according as it was obtained from the evaporated ammoniacal solution or precipitated from its alkaline solutions by acetic acid. In the former case the solubility in hot water was, in round numbers, 1 in 1350; but in the latter, 1 in 396. Prolonged boiling was found by Strecker to lessen the solubility of xanthine in hot water.[3]

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[1] Journal of Chemical Society, Feb. 1862, p. 79. It may be remarked that in no previous account of xanthine have *crystals* of that substance been found. It is to be wished that in Dr. B. Jones's case the identification of xanthine had been more perfect.

[2] Strecker, Liebig's Ann., May, 1861, Bd. cxviii. p. 158.

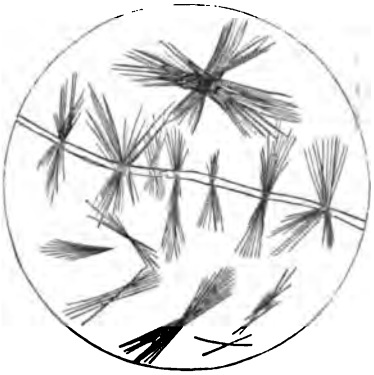
[3] *Ibid.*, p. 168. *See*, also, a recent paper "Sur la Xanthine et sa récherche dans les calculs vesicaux." Lebon. Comptes Rendus, lxxiii. 47.

VIII.—LEUCINE AND TYROSINE.

These two substances were found by Städeler and Frerichs in the urine in typhoid fever and acute yellow atrophy of the liver. Tyrosine has even been found to form a natural urinary deposit in the latter disease. This deposit is described by Frerichs as a greenish-yellow crystalline sediment, which increases considerably with slight evaporation of the urine. Under the microscope, greenish-yellow globular masses, composed of acicular crystals, are seen. In one of Frerichs's cases of acute yellow atrophy, he says of the urine: "After standing in the cold air, a greenish-yellow light sediment was deposited, consisting entirely of acicular crystals of tyrosine aggregated together in globular masses. When a drop of urine was evaporated on a watch-glass, it left behind a residuum, which, upon microscopical examination, was found to be almost exclusively composed of the most characteristic possible crystals of leucine and tyrosine, partly saturated with coloring matter."[1] Frerichs regards the occurrence of these deposits as of great importance in the diagnosis of acute yellow atrophy of the liver.

In May, 1865, my then clinical assistant, Mr. Clements, brought me a specimen of urine passed by a young woman who was suffering (and died the day atter) from acute yellow atrophy of the liver, in the home district of the Manchester Infirmary. After standing forty-eight hours, it had deposited an abundant sediment of tyrosine, crystallized in sheaf-like bundles of acicular crystals (*see* Fig. 16).

FIG. 16.



Tyrosine, spontaneously deposited from the urine of a patient with acute yellow atrophy of the liver.

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[1] Frerichs on Dis. of Liver, Syd. Soc Trans., vol. i., Frontispiece, Fig. 5, and p. 220.

Fränkel[1] found leucine and tyrosine in the urine in cases of poisoning by phosphorus.

Xanthine, *hypoxanthine*, *guanine*, *tyrosine*, *leucine*, *creatine*, and *creatinine*, may be all regarded as intermediate steps in the regressive metamorphosis of azotized tissues of which the ultimate stages are urea, uric acid, water, and carbonic acid. It is therefore not surprising that they should be found in small quantities in the tissues and the blood; and that a retardation of this metamorphosis in some particular stage should occasion their appearance in the urine.

Hitherto their clinical significance has not been made out with sufficient clearness to be of practical service; and the circumstance that (except xanthine and tyrosine) they never form spontaneous urinary deposits, removes them (at present) from the interest and view of the practitioner. But it is not improbable that the study of these bodies in the urine may hereafter lead to important clinical indications; until then, it is not desirable to load a practical work like the present with details respecting them.[2]

IX.—PHOSPHORIC ACID AND THE PHOSPHATES.

Phosphorus exists in the animal body in large quantities, either oxidized into phosphoric acid, and united with bases so as to form phosphates which pervade the fluids and solids—especially the bones; or unoxidized, and combined with albuminous compounds in some manner not yet understood.

Phosphoric acid passes out of the body partly with the feces and partly with the urine. The diurnal excretion of phosphoric acid by the kidneys varies from 30 to 90 grains. The mean of twenty-five sets of observations collected by Dr. Parkes, was 48.80 grains a day. Two-thirds or three-fourths of this are combined with potash and soda to form soluble phosphates, which do not come under the notice of the practitioner as urinary deposits. The remainder is united with lime and magnesia to form salts, which, though soluble in acid urine, are speedily precipitated when the secretion becomes alkaline, and constitute urinary deposits.

Phosphoric acid is derived in part directly from the food; in part also from the oxidation within the body of the phosphorus of the albuminoid tissues. The hourly excretion of phosphoric acid rises considerably after meals; and the earthy phosphates undergo a proportionately larger increase than the alkaline phosphates. In a series of observations extending over six days, I found that the average hourly separation of the earthy phosphates during the two hours preceding dinner, amounted only to one-half the quantity separated during the third and fourth hours after dinner. The alkaline phosphates rose from 3.47 grains per hour before dinner, to 4.90 grains after dinner.

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[1] Berl. klinisch. Wochensch., 1878, p 265.

[2] A paper, by K. B. Hoffmann, in Virchow's Archiv, Bd. 48, p. 358, may be referred to as containing a large amount of information respecting the excretion of creatinine in health and disease.

The food is, however, not the sole source of the phosphoric acid of the urine; and the separation of it goes on, though in greatly diminished quantity, after prolonged fasting.

A very large number of observations has been made on the excretion of phosphoric acid in disease, but with results of slight clinical value. Dr. Bence Jones has formulated the following conclusions (founded on determinations per 1000 parts): "In acute inflammation of the brain, there is an excessive amount of phosphates in the urine. When the inflammation becomes chronic, no excess of phosphates can be shown to exist. . . . In some functional diseases of the brain, an excessive amount of phosphates is observable; this ceases with the delirium. Delirium tremens shows a remarkable deficiency in the amount of phosphates excreted, provided no food is taken. When food is taken the diminution is not apparent."[1] These observations are substantially borne out by the observations of Tomowitz and Beale.

Professor Vogel ascertained the rate of excretion of phosphoric acid in a very great number of acute and chronic diseases (having made above 1000 observations), but without eliciting any conclusions capable of clinical use.[2]

Zülzer[3] again has determined the amount of phosphoric acid in the urine in relation to the nitrogen excreted. He finds that in ordinary health the "relative amount" of phosphates is fairly constant although changed somewhat by food, as mentioned above. In depressed states of the system, however, phosphates are excreted in excess, while in excited states they are diminished. In the febrile state again the nitrogen of the urine is increased, but the phosphates are diminished. When defervescence sets in, large quantities of both are eliminated, and during convalescence the phosphates predominate. The condition of the urine in cholera was found to be peculiar. That passed in the early stages was rich in phosphates, while as the patient recovered the phosphates again diminished. Zülzer also found an increased excretion of phosphates after injuries to the nervous system.

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[1] Medico-Chir. Trans., vol. xxxviii, p. 261.

[2] Neubauer and Vogel: Analysis of the Urine, Syd. Soc. Trans. p. 413—where the reader is referred for fuller information. Dr. Paton found that ment work caused no increase, but rather a diminution, of the phosphates in the urine. Journ. of Anat and Phys., May, 1871.

[3] Virch. Arch., Bd. 66, p. 223.

Salkowski,[1] however, objects that in Zülzer's conclusions sufficient attention was not paid to the facts that:

1. Phosphates are excreted in the feces as well as in the urine.
2. That the character of the food has great influence on the excretion.
3. That the bones are rich in phosphoric acid, and they possibly take some part in the metabolic changes which occur in the body.

To the practitioner, therefore, the interest of phosphoric acid and the phosphates in the urine, is confined to the earthy phosphates which come before him as urinary deposits and urinary concretions.

Dr. Prout dignified with the name of "phosphatic diathesis," the tendency to the deposition of the earthy phosphates in the urine. Dr. Bence Jones[2] has, however, clearly shown that this designation is wholly inappropriate. There is not the least reason to believe that there is any constitutional state specially characterized by an excessive excretion of phosphates; the phosphatic diathesis of Prout is simply ammoniacal urine.[3]

DEPOSITS OF EARTHY PHOSPHATES.

Phosphoric acid is spontaneously deposited in the urine chiefly, if not exclusively, in one of the three following combinations:

1. Amorphous phosphate of lime, or bone-earth ($Ca_3(PO_4)_2$).
2. Crystallized phosphate of lime ($CaHPO_4+2Aq.$).
3. Ammoniaco-magnesian phosphate, or triple phosphate ($MgNH_4PO_4+6Aq.$).

These three compounds are occasionally precipitated together in one deposit; much more frequently the first and third are found together, forming the ordinary sediment of ammoniacal urine. This latter passes under various names, viz.: "the mixed phosphates," the "secondary phosphates," or "fusible matter." This will come under notice again as the special constituent of secondary calculous formations.

The earthy phosphates are readily soluble in the natural acid of the urine; but are insoluble in neutral or alkaline fluids.

Their precipitation as deposits is, therefore, properly associated with an alkaline state of the urine; it is, nevertheless, a fact that the second and third forms are occasionally deposited in urines that are neutral or feebly acid.

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[1] Die Lehre vom Harn, p. 186.

[2] Animal Chemistry, p. 85.

[3] (Teissier, of Lyons, has described certain cases under the title of "Phosphatic Diabetes," which are characterized by polyuria with excessive excretion of phosphates, and many of the symptoms of ordinary diabetes. Dr. Ralfe (Lancet, Jany. 1881, p. 406) has also called attention to, and described such cases. The affection, however, seems to have great affinities with Diabetes Insipidus.—R. M.)

Urines depositing the earthy phosphates, or tending thereto by their neutral or feebly acid reaction, become turbid when heated. This behavior has been variously explained. Some have thought that the heat expelled the carbonic acid which held the earthy phosphates in solution; others, that the heat caused rapid decomposition of the urea into carbonate of ammonia, and thereby suddenly increased the alkalescence of the urine. Scherer thought the reaction due to the conversion of the neutral phosphates of lime and magnesia into basic salts. I think that a more simple explanation may be offered. Some salts of lime (like the hydrate) are much more soluble in the cold than at higher temperatures. This is probably the case with the lime phosphates. I find that when a urine which exhibits this reaction is heated in a sealed glass tube by immersion in hot water, the turbidity speedily makes its appearance; but it disappears slowly (either wholly or partially) after the tube has cooled. On reheating the turbidity returns, and again slowly disappears after cooling. This experiment may be repeated many times with a similar result. The same succession of events may be obtained by heating the urine in an ordinary test-tube over the open flame, provided the heating be not pushed to ebullition. If such a urine be sharply boiled there is no resolution of the precipitate after cooling, because probably the chemical change suggested by Scherer has been brought about.[1]

1.—*Amorphous Phosphate of Lime, or Bone-earth.*

This compound is invariably precipitated in alkaline urine. When the urine is alkaline from *fixed* alkali, this is the ordinary, and often the sole deposit; but far more frequently it is accompanied by the triple phosphate.

It forms an amorphous, whitish, light flocculent deposit, indistinguishable by the naked eye from epithelium. It has no affinity for the coloring matter of the urine, and is consequently of a paler color than the supernatant urine, differing in this respect from the amorphous urates. The surface of the urine is generally covered with an iridescent film.

The application of heat does not dissolve the deposit, but, on the contrary, increases it. A drop of any acid causes it instantly to disappear. Under the microscope it appears as very pale, minute granules in irregular clumps or patches, much resembling the fawn-colored lithates (*see* Fig. 3).

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[1] For another explanation of this reaction, see a paper by Prof. Smith in the Dublin Journ. of Med. Sci., July, 1883.

Its occurrence depends simply on the existence of an alkaline reaction, and the presence of lime and phosphoric acid in the urine.

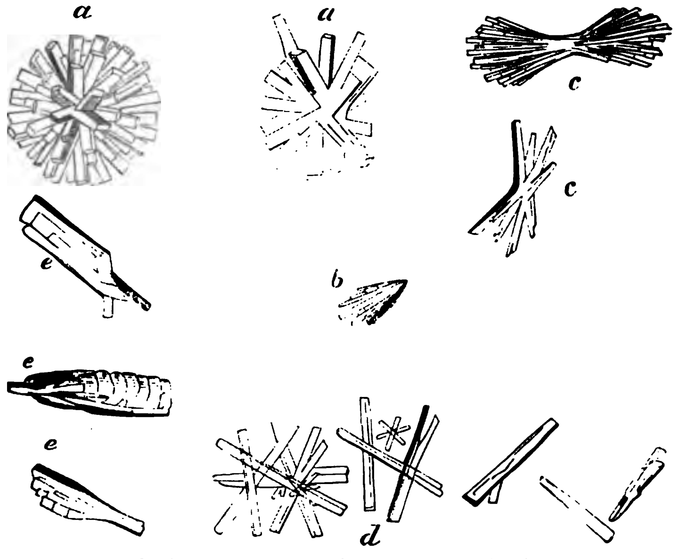
This is the normal deposit of the alkaline urine after a meal. It is also frequently seen in persons whose urine has been rendered alkaline by remedies (carbonates, acetates, citrates of the alkalies, etc.), and after the excessive use of sweet and subacid fruits. The turbidity caused by the amorphous phosphate exists in its greatest intensity at the moment of emission of the urine, and does not increase on cooling.

The clinical significance and treatment of this deposit are entirely involved in those of alkaline urine. Bone-earth alone very rarely constitutes a urinary calculus; but it enters largely into the composition of phosphatic calculi in combination with the ammoniaco-magnesian phosphate.

2.—*Crystallized Phosphate of Lime, or Stellar Phosphate.*

Dr. Hassall first called attention to the existence of a crystallized form of phosphate of lime occurring as a urinary deposit.

FIG. 17.



Stars and rods of crystallized phosphate of lime, or stellar phosphate.

In 1860 he communicated a paper to the Royal Society on the composition and pathological importance of the calcareous phosphates occurring in the urine as a spontaneous deposit of stellar crystals. He considered these crystals to consist of biphosphate of lime; he also believed them of far graver significance than the triple phosphate of ammonia and magnesia.

In 1861 I had an opportunity of reëxamining this question, and published the results of my observations in the "British Medical Journal" for March 30, 1861.

The crystals in question present considerable variety of form (Fig. 17).

The prevailing appearance is that of crystalline rods or needles, either lying loose (*d*), or grouped into stars, rosettes (*a a*), fans (*b*), or sheaf-like bundles (*c*). Some of the crystals are club or bottle-shaped (*e e*), and abundantly marked with lines of secondary crystallization.

In a case of diabetes under my care in the Manchester Infirmary, these crystals formed a constant deposit. The urine had been brought down by appropriate treatment to fifty ounces a day, and the patient was steadily gaining flesh and strength. The deposit was often mixed with oxalate of lime, and sometimes with uric acid; but never, except as the result of putrefactive decomposition, with the triple phosphate. I managed to collect about two grains of the crystals in a pure state, and subjected them to analysis. The results indicated the following formula: $2CaO,HO,PO_5+3HO$ (old notation).

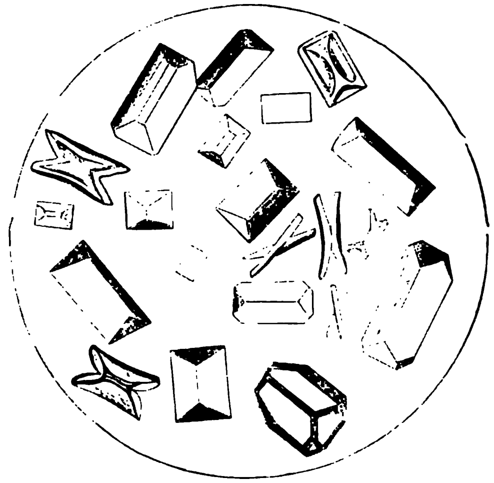
By adding a little chloride of calcium to healthy urine, and reducing its acidity to near the neutral point with caustic soda, I have often succeeded in obtaining an abundance of crystals closely resembling those occurring spontaneously in urine. The reaction of the urine in which I have found the crystallized phosphate of lime has been sometimes faintly acid, more often neutral, or alkaline.

The occurrence of a deposit of the stellar phosphate in urine is not common. It is, in fact, a rare deposit, as compared with oxalate of lime, uric acid, or the triple phosphate. The presence of this deposit in quantity is, according to my experience, an accompaniment of some grave disorder. In addition to the case of diabetes already mentioned, I have seen the stellar phosphates in cancer of the pylorus, once in phthisis, and more than once in patients exhausted by obstinate chronic rheumatism. They may, however, under peculiar conditions, be precipitated in a healthy urine. When the urine is rich in lime, and its acidity is at the same time depressed to near the neutral line, stellæ of phosphate of lime may form quite independently of any grave disorder, merely as the result of a coincidence in the chemical composition and reaction of the urine. For example, after a full meal the acidity of the urine becomes greatly reduced, and lime derived from the food is in excessive proportion. In such circumstances, I have several times detected stellæ of phosphate of lime, but only in scanty numbers. A depressed acidity of the urine is an essential contingent to the formation of these crystals; and if the urine subsequently to their formation increase in acidity, they may spontaneously disappear.

3.—*The Phosphate of Ammonia and Magnesia, or Triple Phosphate.*

This is an insoluble crystalline compound, which occurs very frequently as a urinary deposit—sometimes alone, but much more commonly accompanied with the amorphous phosphate of lime. When unmixed with any other substance, the deposit has a snow-white appearance; and bright, sparkling, colorless crystals are observed studding the sides of the urine-glass and forming a brilliant crystalline film on the top. The ordinary form of the crystals is a triangular prism with bevelled ends. A very great variety of subordinate forms is produced by a planing off of the ridges and angles, and a hollowing out of the sides (Fig. 18). In a highly ammoniacal urine, the magnesian phosphate forms elegant dielytral crystals, which appear to arise from a hollowing of the sides and a deep notching of the extremities of the prisms.

FIG. 18.



Different forms of triple phosphate crystals.

The triple phosphate is easily soluble in acids; yet it may be found in urine that is feebly acid to test paper. Heat does not affect it; and the urine which deposits it commonly becomes turbid on boiling.

This deposit is necessarily present in ammoniacal urine, except in the very rare contingency of the urine not containing any magnesia. When urine is alkaline from fixed alkali, crystals of this salt generally appear after a while. This is easily explicable after the demonstration by Neubauer and Heintz that ammoniacal compounds exist in small quantities even in fresh natural urine.

In the immense majority of cases the deposition of this salt is only an incident due to the loss of the acid reaction of the urine, and especially of ammoniacal decomposition of the urine. Occasionally, however, it occurs in fresh urine which is neither decomposed nor sensibly (to the smell) ammoniacal. The following is the most remarkable instance which I have witnessed: J. P., a gentleman, aged twenty-nine, of a moderately healthy appearance, but irritable temperament, consulted me on account of a sense of weakness in the back and loins, with general debility and languor, and a tendency to sudden perspirations and fits of nervousness. There was severe smarting at the close of micturition. He had suffered from gonorrhœa three years previously, but had been completely free from any urethral discharge for some time. The urine was examined on several occasions. It was faintly acid when voided; and deposited, sometimes before it was cold, and generally within a couple of hours, an abundant precipitate of the unmixed ammoniaco-magnesian phosphate. The annexed note was taken of the urine voided at 11.30 A. M. on January 28, 1861. "In half an hour it was found transparent, perfectly sweet (*i. e.*, not putrescent), faintly acid; and sparkling crystals of the triple phosphate could be seen floating in it. At four P. M. the same day the specimen was quite clear; brilliant crystals of triple phosphate studded the sides of the glass, and at the bottom was collected an abundant snow-white deposit of the same crystals. The urine was not albuminous, neither did it contain pus or epithelium. On the following day the specimen continued unchanged; but on the fourth day the reaction had become faintly alkaline; the deposit was losing its snow-white character, and reddish flakes, composed of spheres of urate of ammonia, had become deposited. From this date the urine began to decompose, and speedily became ammoniacal and offensive." This condition of the urine, together with the unpleasant symptoms before noted, gradually disappeared in the course of six weeks, under the influence of cold sponging, systematic exercise in the open air, and the administration of dilute nitric acid in a bitter infusion.

Stein[1] found a deposit of magnesium phosphate, $Mg_3(PO_4)_2$, in the strongly alkaline urine of a patient suffering from dilatation of the stomach.

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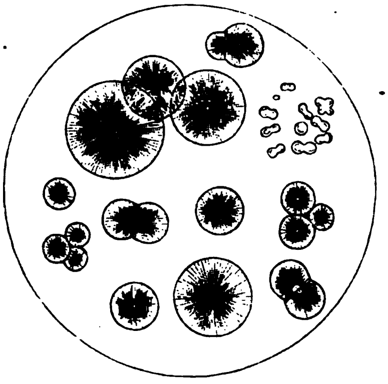
[1] Deutsch. Arch. f. klin. Med., Bd. 18, p. 207.

X.—CARBONATE OF LIME.

When urine becomes alkaline from carbonate of ammonia, a small quantity of carbonate of lime is precipitated in an amorphous condition with the earthy phosphates. I have only seen it in a crystalline form in human urine when voided as gravel or small calculi; it is said occasionally to occur in globular spheres and cornucopia-like crystals (Bird, Hassall). In the alkaline and viscid urine of the horse, carbonate of lime is frequently deposited in the form of minute spheres composed of radiating linear crystals which are striking objects under the microscope (*see* Fig. 19). They show a dark cross with polarized light. The assumption of this globular form is probably connected with the viscidity of the urine.

Fig. 19.



Spheres and dumb-bells of carbonate of lime from the urine of the horse.

Carbonate of lime constitutes a variety of urinary calculus which is of extreme rarity in the human subject, but much more common in the herbivora (*see* Carbonate of Lime Calculi).

XI.—SULPHURIC ACID AND THE SULPHATES.

About thirty grains of sulphuric acid, in combination with alkaline bases, are daily excreted by the kidneys. A part is derived directly from the food, and a part from the oxidation of the sulphur contained in the albuminous compounds. The sulphates are highly soluble, and in only two cases have they been known to constitute a spontaneous urinary deposit.[1]

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[1] Valentiner, Centralblat. f. Med. Wissen, 1863, p. 913; Fürbringer, Deutsches Arch. d. klin. Med., Bd. 20, p. 511.

In all febrile states sulphuric acid is increased. Dr. Parkes has observed a decided increase after the use of liquor potassæ. An increase is also observed after food, and in all conditions associated with an intensified metamorphosis of tissue.

It has not yet been shown that a knowledge of the quantity of sulphuric acid separated by the kidneys in any particular case of disease is capable of subserving any practical purpose.[1]

XII.—CHLORINE AND THE CHLORIDES.

The chlorides never form spontaneous deposits in the urine; and the variations in their quantities have only an uncertain relation to special states of disease, but depend chiefly on the times of the meals and on the general rate of tissue-changes.

A good deal of attention has been called to the falling off or disappearance of the chlorides in the urine in acute pneumonia, and their reappearance when resolution is established. It has been asserted that a knowledge of the amount of chlorides excreted by the kidneys in the course of this disease, furnished valuable information for prognosis and treatment. Later observations have, however, shown that the indication is far from being a reliable one, and that the notions entertained in some quarters of its utility are greatly exaggerated. Although it be a rule of very prevalent application, that the chlorides in the increment stage of acute pneumonia are almost completely retained within the body, and that their reappearance in the urine is coincident with commencing resolution, yet there are exceptions to both these statements, especially to the coincidence of the reappearance of the chlorides with commencing defervescence (see Parkes).

SUPPLEMENTARY REMARKS ON THE EXCRETION OF PHOSPHORUS, SULPHUR, AND CHLORINE.

These three elements enter largely into the composition of the body, and they are abundantly present in articles of food. They pass out of the body chiefly with the urine; but partly also with the feces. Multiplied observations have been made, and continue to be made, on the rate of their excretion both in health and disease; and important physiological and pathological deductions have been drawn from these investigations.

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[1] [In certain rare cases sulphuretted hydrogen may appear in the urine. In most of such cases there has been found either some communication between the intestinal and urinary tracts, or a pericæcal abscess from which the gas was absorbed. Occasionally, however, neither of these conditions seems to have been present, and the origin of the phenomenon was obscure. *See* a case by Cameron, Lancet, 1880, vol. ii. p. 766. R. M.]

It has been considered that the rate of excretion of phosphorus and sulphur, under proper precautions and corrections, furnished a measure of the exchange of material within the body—that is, of the activity of the molecular life of the tissues: and that in disease, an important insight into obscure phenomena could be thus obtained, capable of being turned to practical uses. In proportion, however, as these researches have been extended, it has become clearer and clearer that these expectations are not likely to be realized, and that the practitioner is not likely to draw much help from these recondite sources. The difficulties in the way are manifold. In the first place, quantitative determinations of sulphur and phosphorus, notwithstanding all the aid of modern volumetrical methods, are still too troublesome and tedious to be within reach of any but a very select body of practitioners. But this is one of the smallest difficulties. In all such determinations it is necessary to do more than ascertain the proportion per cent. To obtain results of any value, the quantity per day must be ascertained. Again, there are physiological variations to make allowance for, arising from food, exercise, sleep, etc.; and thirdly, it has now been ascertained that, all known conditions remaining the same, the rate of excretion of these elements presents oscillations from an unexplained temporary retention, or partial retention, of the elements within the body, which is succeeded, after a shorter or longer interval, by a compensating increased discharge. These circumstances render it necessary to continue the observations over a number of days—six or eight—in order to cover the inequalities. For these and other reasons which might be mentioned, these inquiries are surrounded with difficulties. It is little wonderful, therefore, that the results obtained by different experimenters show a marked want of uniformity: and it is simply the fact that, from a clinical point of view, these laborious investigations must at present be regarded as unfruitful, and for that reason they may be passed over with only a slight notice in a practical work. It is highly desirable, however, that researches of this class should be pushed on; it is impossible to say how soon practical lessons may be culled from these now apparently dormant facts. At any rate, they cannot fail to enlarge our general ideas on physiological and pathological processes.

XIII.—UREA, $CO(NH_2)_2$.

Looking at the urine from a physiological point of view, urea must be regarded as its most important constituent. It is the chief final product of the metamorphosis of the albuminous tissues, and furnishes the form under which nearly all the nitrogen finds its way out of the body.

Urea is a bland crystalline substance possessing the properties of a feeble base. Its best known combinations are the nitrate and oxalate, both of which are much less soluble than urea itself. Urea is very soluble both in water and alcohol; it never forms a spontaneous urinary deposit. Its presence in a urine of high density, or one artificially concentrated, is easily demonstrated. If to such a urine an equal volume of strong nitric acid be added, in a test-tube, and the tube be plunged into cold water, the mixture speedily becomes a shining mass of crystals of nitrate of urea.

The daily separation of urea by adult men between the ages of twenty and forty, averages about 500 grains; but the amount varies considerably from various causes, such as diet, exercise, meteorological conditions, and individual peculiarities. Of the twenty-four series of observations, of not less than six days each, tabulated by Dr. Parkes, the minimum result is 286.1 grains and the maximum 688.4 grains per day. The body-weight has, as might have been expected, a very apparent relation to the daily excretion of urea, but the relation is not simply a direct one, because the weight of individuals is made up differently—some being heavy from bone and muscle, others from an accumulation of fat. It is estimated that a healthy adult man excretes urea at the daily rate of 3½ grains per pound of the weight of his body.

The excretion of urea is greatly increased after a meal—especially of animal food. Bidder and Schmidt believed that this arose from a direct transformation into urea of a portion of the alimentary materials without their being previously fixed as tissues; but Bischoff and Voit, with more probability, attribute this increase to an accelerated tissue-metamorphosis induced by the presence of the new supplies in the blood.

Copious water-drinking causes an increased separation of urea. Children secrete more in proportion to their weight than adults.

The immediate effect of muscular exercise appears to be to restrain (or at least not to increase) the excretion of urea; but it is increased in the period of rest which follows exercise.[1]

The quantitative estimation of urea in urine may be made either by the method of Liebig or by that of Davy, as modified by Drs. Russell and West.

Liebig's Volumetrical Method.—This method is based on the property of urea to form an insoluble precipitate of fixed composition with the nitrate of the protoxide of mercury. But in order that the test may operate, it is necessary to free the urine beforehand from phosphates and sulphates. It is also necessary for complete accuracy to make allowance for the chloride of sodium present. When chloride of sodium coexists in any fluid with urea, the nitrate of mercury produces no precipitation of urea until the whole of the chloride of sodium is decomposed with formation of bichloride of mercury and nitrate of soda. After this conversion is completed, urea begins to be precipitated, and the test-solution is to be added until no more urea remains in solution. This point is ascertained by a solution of carbonate of soda, which immediately develops a yellow color when—and not before—all the urea has been thrown down with the mercury.

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[1] For further information respecting the excretion of nitrogen (urea) under various conditions, the reader is referred to the important investigations of Dr. Parkes, Med. Times and Gaz., 1867, I. 393, and Proc. Roy. Soc., 1871, p. 349; also Grehaut, J. de l'Anat. et Phys., 1870, 318; Weigelin, Reichert's Archiv, 1868, 207; Paton, Journ. of Anat. and Phys., May, 1871.

Three solutions are therefore required.

First. A baryta solution, to precipitate the phosphates and sulphates. This is composed of one volume of a cold saturated solution of nitrate of baryta mixed with two volumes of saturated baryta-water.

Second. The mercurial test-solution. Ten cubic centimetres of this solution contain 0.772 gramme of red oxide of mercury dissolved in nitric acid—that is to say, in the form of nitrate of the peroxide of mercury.

Third. A solution of carbonate of soda of about the strength of twenty grains to the ounce.

As the preparation of the first and second solutions is very troublesome—the latter especially—it is more convenient to purchase them ready made.[1]

The analysis is performed in the following manner:

1. Forty cubic centimetres (or two volumes) of the urine are mixed in a beaker with twenty cubic centimetres (one volume) of the baryta solution. The mixture is thrown on a filter; fifteen cubic centimetres of the filtered fluid (which, of course, contains two-thirds, or ten cubic centimetres of urine) are carefully measured off and placed in a small beaker.

2. A graduated burette is filled with the mercurial solution, which is then very carefully dropped into the beaker until the mixture begins to become turbid; a few drops generally suffice. A note is taken of the quantity of the solution used to reach this point: it indicates that all the chloride of sodium is decomposed and that the urea is now beginning to be precipitated.

3. The mercurial solution is now added more freely, and thoroughly mixed by means of a glass rod: a copious white

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[1] These and other test-solutions for volumetrical analyses of the urine may be had of Griffin, Bunhill Row, London, and from Mottershead & Co., Chemists, Manchester.

precipitate makes its appearance, and the analysis approaches completion.

4. This point is ascertained by pouring some of the carbonate of soda solution into the bottom of a white porcelain plate; and taking a drop from the turbid mixture in the beaker by means of the stirring-rod, and letting it fall into the solution on the plate. As long as the drop produces only a white curdy circle the mercurial solution is still to be added; but as soon as a yellow tinge appears the analysis is finished.

5. The quantity of mercurial solution used is then read off, and the portion used before the occurrence of turbidity subtracted—the remainder is what has been employed to precipitate the urea. Each cubic centimetre of the solution used indicates 0.154 grain (or 0.01 gramme) of urea. From this, by an easy calculation, the amount of urea in ten cubic centimetres of urine may be ascertained; and if the number of cubic centimetres of urine voided in the twenty-four hours be known, the daily excretion of urea is readily calculated.

DAVY'S PROCESS MODIFIED BY RUSSELL AND WEST.—The principle of this method depends on the decomposition of urea by the hypochlorites and hypobromites. The amount of urea is determined by measuring the volume of nitrogen evolved.

A solution is prepared by dissolving 100 grammes of solid caustic soda in 250 c.c. of water and adding 25 c.c. of bromine at the time the solution is required.[1] The apparatus constructed by Drs. Russell and West for the performance of the analysis is both compact and cheap.[2] But it is not so convenient and accurate to work with as that devised by Mr. Apjohn. Mr. Apjohn's apparatus consists of:

1. A glass measuring tube of about a foot in length drawn out at the end which will be uppermost when the tube is used, like a Mohr's burette, and subdivided into 30 parts of equal capacity, the aggregate volume of which is 55 c.c.
2. A small wide-mouthed gas bottle of about 60 c.c. capacity.
3. A short test-tube of about 10 c.c. capacity, and of such height that when introduced into the gas bottle it will stand within it in a slightly inclined position.

The following are the arrangements for combining the apparatus and working an experiment:

The graduated tube, held in a clamp attached to a retort-stand, is depressed into a glass cylinder, nearly filled with water, until the zero mark, which is near the upper end, exactly coincides with the surface of the water. 15 c.c. of the hypobromite solution (100 grammes of NaHO, 250 c.c. of water, 25 c.c. of bromine) having been poured into the bottle, the test-tube containing the urine is introduced by means of a forceps, care being taken that none of its contents shall spill into the hypobromite. The flask is now closed with a very accurately fitting India-rubber stopper, perforated with a hole in which is inserted a short piece of glass tubing open at both ends, and is then connected with the measuring tube by means of a piece of elastic tubing. It is now inclined so as to allow the urine to mix with the hypobromite. Effervescence at once commences, and as it proceeds the measuring tube is gradually raised so as to relieve the disengaged nitrogen from the hydrostatic pressure. The flask is shaken a few times, and when the reaction is completely over, the apparatus is left for a few minutes until it has acquired the temperature of the room in which the experiment is performed. Another exact levelling of the measuring tube is made, and the number of the division corresponding to the volume of the developed nitrogen is read off.

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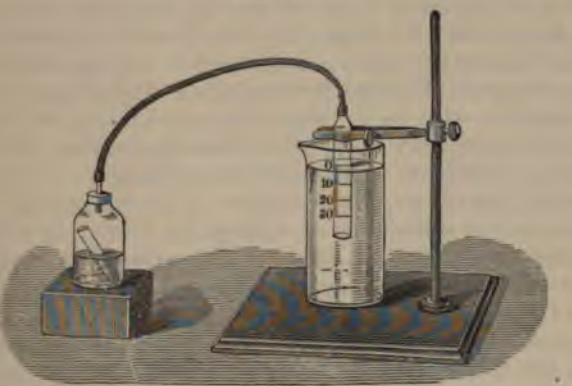
[1] Lancet, February, 1876, p 241.

[2] It may be had from Cetti, Brooke Street, Holborn, London, price 8s 6d.

FIG. 20.



Apjohn's apparatus for the estimation of urea with Russell and West's solution.

The tube is so graduated that, when 5 c.c. of urine are operated on, each division corresponds to 0.1 per cent. of urea, or 0.44 grain per fluidounce of the British Pharmacopœia. An easy calculation from these data gives the daily discharge of urea. Suppose 45 ounces of urine are voided in the 24 hours, and that 5 c.c. of this evolve 20 measures of nitrogen with the hypobromite solution, then:

$$0.44 \times 20 \times 45 = 396.$$

The daily discharge of urea was 396 grains.

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I have carefully tested this method and have found it easy, accurate, and speedy.[1]

PATHOLOGICAL RELATIONS OF UREA.—The excretion of urea in disease has been examined in a large number of cases. In the acute stage of febrile and inflammatory diseases, there is an increased formation and discharge of urea, depending on an accelerated metamorphosis of tissue. When the crisis of the disorder has passed, and defervescence sets in, the excretion of urea falls even below its natural average. This rule, however, is liable to exceptions; it appears that in not a few instances there is a retention of urea within the body during the pyrexial period, even when no disorder of the kidneys exists, and a compensating discharge when convalescence begins. Acute (inflammatory) Bright's Disease is a constant exception; the urine in that complaint is poor in urea; but this arises, not from diminished formation, but from defective separation, owing to the blocked-up condition of the uriniferous tubes.

Frerichs found, in one example of acute yellow atrophy of the liver, a total deficiency of urea in the urine; in a second case there was abundance of urea in the urine discharged during life, but only a trace in that withdrawn from the bladder after death.

In chronic diseases not involving the kidneys, the excretion of urea has not usually been found materially affected.

In saccharine diabetes there is an excessive separation of urea, as might have been expected from the accelerated rate of tissue metamorphosis which must accompany the full feeding and rapid emaciation of these patients.

In a case of diabetes insipidus (with a daily discharge of 12 or 14 pints of urine), I found the excretion of urea to oscillate between 394 and 505 grains daily, which yielded a mean rate of 4½ grains per pound of the body-weight. This is about a fourth above the average for healthy individuals.

In both acute and chronic degeneration of the kidneys (Bright's Disease) there is a marked lessening of the excretion of urea, as will be more fully commented on when those diseases come to be described. The proportion of urea is also greatly reduced in the urine voided by persons suffering from an obstruction in the ureters (*see* Suppression of Urine).

One of the most important properties of urea is the great facility with which it is broken up and resolved into new compounds. This property comes into important play when urea is unnaturally retained in the blood or in the urinary passages.

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[1] For a further account of this method, *see* Russell and West's paper, Journ. of Chem. Soc., Aug. 1874; and Practitioner, Feb. 1875. Apjohn's paper is in the Chemical News, Jan 22, 1875.

It has been already explained with what consequences this bland and innocuous base is converted into pungent carbonate of ammonia in the bladder and other parts of the urinary tract. A similar conversion, taking place in the blood, was believed by Frerichs to be the cause of the stormy and dangerous phenomena of uræmia.

Dr. Prout believed that there existed a peculiar morbid state characterized by an absolute and relative increase of the excretion of urea, unaccompanied by pyrexia. To this condition Dr. Willis, who adopted the view of Prout, gave the name of *Azoturia*. The subjects of this form of disease, according to Prout, had usually a frequent and urgent desire to pass water both by night and day. This seemed principally due to an irritable sensation referred to the neck of the bladder, occasionally extending along the urethra; but in some cases it was due, at least in part, to real diuresis. In almost every instance the quantity of urine voided in the twenty-four hours was somewhat above the natural standard. The quantity was also particularly liable to be increased by causes which would scarcely affect a person in perfect health, at least to the same degree; such as by a chilly state, mental emotion or excitement, etc.[1]

In addition to the direct urinary symptoms, there was sometimes a sense of weight or dull pain in the back, accompanied by disinclination to bodily exertion; there was no remarkable thirst; nor craving for food; nor emaciation. Moreover, the functions of the skin appear to be little deranged.

Such is a summary of the description of Prout. He does not supply any details as to the daily flow of urine nor the daily amount of urea. At the time Prout wrote, very little was known as to the natural (physiological) variations in the excretion of urea; and the opinion he held as to urea being chiefly the final product of the metamorphosis of the gelatinous tissues has since been proved to be erroneous. Looking at the question from the standing-point of the physiological doctrines now in the ascendant, it is difficult to admit the existence of a condition characterized by the incompatible coincidences of an increased excretion of urea, with absence of thirst, absence of excessive feeding, and absence of emaciation.

Precise facts in support of Prout's view are wanting. Willis's description is too loose to give much confidence, and subsequent writers have contented themselves with a reference to Prout and Willis.[2]

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[1] Prout: Stomach and Renal Diseases, 5th edit., p. 97

[2] The six cases recorded by Dr. Handfield Jones, in the Brit. Med. Journ. for Oct. 12, 1861, under the title of "Cases of Baruria," are so deficient in necessary details, that they are of no service to a reader. In only one of them was the urine of the twenty-four hours collected and examined, and in that case only on one occasion. In the remainder "baruria" seems to have been inferred to exist from the high density of a single specimen.

Dr. Parkes,[1] however, records a remarkable case examined by Dr. Ringer. The patient was a middle-aged man weighing 109 pounds, who was not feverish, and appeared only feeble. He was fed on the ordinary diet of the hospital (University College), and passed in each twenty-four hours no less than 1130 grains of urea (mean of twelve days), or 10.36 grains to each pound avoirdupois of his body-weight. There was a trace of sugar, but not enough to determine quantitatively. The daily flow of urine in this case amounted to 96 fluidounces, which is fully double the normal average.

In my own experience, I have usually found that cases which at first sight appeared to belong to this category—cases exhibiting a dense urine and a train of nervous symptoms—turned out on more exact investigation to want the special feature indicated by Prout as the essential one; namely, an absolute increase in the daily discharge of urea. Nevertheless, some facts, rarely observed, have left an impression on my mind that Prout's description is not altogether fanciful. The following case, which I saw with the late Mr. Greaves, of this town, seems to have been one of those Prout had in view when he drew up his account.

Mr. L., a man about 50, complained of troublesome irritation at the back of the pharynx, debility, want of energy and power of application to business. In the preceding three months he had lost 20 lbs. in weight.

The urine was first examined by me on May 23, 1863. It had sp. gr. 1029, and contained a small quantity of sugar, but less than one grain to the fluidounce. This was the only occasion on which I detected sugar, but Mr. Greaves had found it once or twice previously. It was arranged that the whole of the urine voided in each 24 hours should be separately collected and sent to me for analysis. This was done for three successive days; and three weeks later it was done again for two successive days. The following table exhibits the result of the examination:

	Quantity per day.	Sp. gr.	Total urea.
May 25. . . .	27 ounces	1029.5	542 grains.
" 26. . . .	30½ "	1029.75	559 "
" 27. . . .	31 "	1028.25	555 "
June 18. . . .	29 "	1027.5	565 "
" 19. . . .	34 "	1020.5	510 "

This patient was not febrile; his weight was 120 lbs.; there was little appetite, and no thirst, and yet he excreted daily 4.6 grains of urea for each pound of body-weight on these five days, which is fully a quarter beyond the usual average. I saw the patient again towards the end of January, 1863. The urine had then lost its peculiarity; and the health, under a regulated diet and exercise, and a course of vegetable tonics, with citrate of potash, had become completely reëstablished.

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[1] Parkes: On the Composition of the Urine, p. 374.

Prout was of opinion that these cases were pathologically related to diabetes; and he conjectured, though he had not witnessed the fact, that they often developed subsequently into that disease. That there is some relation between the two conditions seems not improbable; in the cases of Dr. Ringer and myself a small quantity of sugar was temporarily present in the urine with the excess of urea.[1]

In the case just related the cause of the complaint was mental anxiety; and in all the instances which I have been inclined to place in this group, the origin of the disorder could always be traced to some kind of mental emotion.

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[1] See Demange, Thèse de Paris, 1878, and London Medical Record, 1879, p. 93.

CHAPTER IV.

ABNORMAL SUBSTANCES IN THE URINE: ORGANIC DEPOSITS.

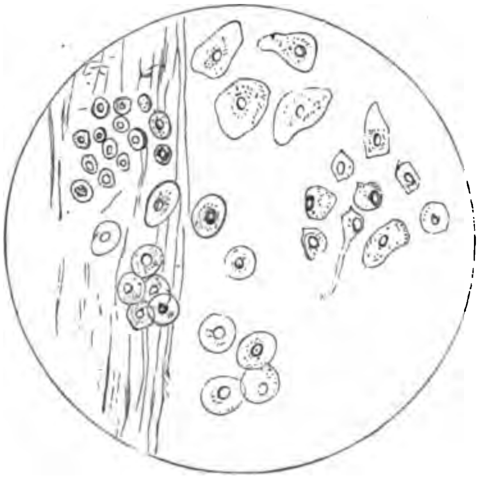
I.—EXTRA-RENAL EPITHELIUM.

ANY part of the genito-urinary passages may shed its epithelium into the urine so as to form a sediment.

The urine of the two sexes differs notably in the character and quantity of the epithelial cells found therein. This arises from anatomical differences in the lower genito-urinary passages; and advantage may sometimes be taken of this circumstance to distinguish the sex of the individual whose urine is under examination.

In the male sex an epithelial deposit of extra-renal source is most commonly derived from the urethra and prostate gland, and is composed of oval, tailed, or rounded cells (Fig. 21), about twice as large as pus cells and usually flattened. A deposit of this sort is always scanty, and to the naked eye presents the

FIG. 21.

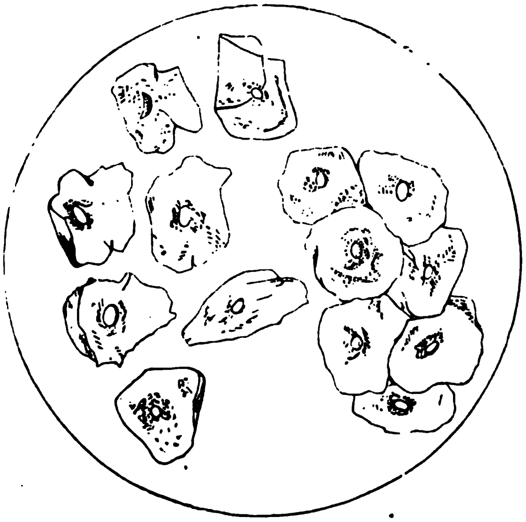


Oval and tailed epithelial cells, found in the thready and flaky deposit of the urine of a man who had formerly suffered from gonorrhœa.

appearance of a collection of whitish flakes and strings. When taken up by the pipette for examination, these flakes are found to have the viscid glairy character of mucus. A sediment of this character is not uncommon in men; in many cases it may be distinctly traced to an old gonorrhœa, which has long since passed away leaving no other vestiges behind it. The deposits found in the urine of persons subject to nocturnal emissions have very much the same appearance to the naked eye.

It is well to be aware of the nature of this deposit. Youths principally, but older men not unfrequently, observe for themselves the presence in their urine of the strings and flakes just described; and they are liable to become subject to hypochondriacal fears and anxiety respecting them. Such individuals are common victims of unprincipled empirics. I was recently consulted by a gentleman who paid very large sums to a quack who had persuaded him that the flaky shreds in his urine—the innocuous vestiges of a gonorrhœa contracted five years previously—were of a dangerous nature, and required active and long-continued treatment. It is not a trifling matter to be able to allay the alarm of such patients, and to convince them that the subject of their anxiety is wholly unimportant.

Fig. 22.



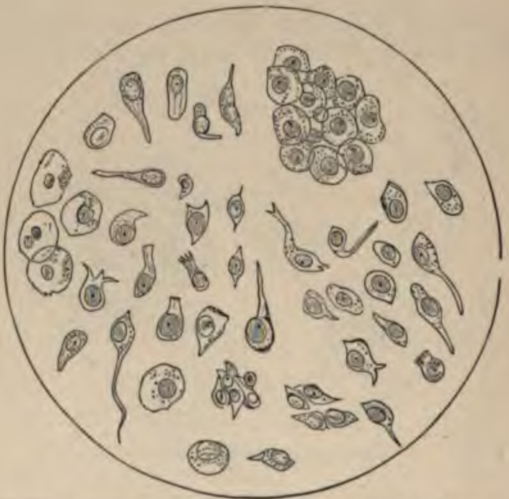
Vaginal epithelium in the urine.

In females, epithelial sediments are both common and abundant. From the simple short urethra the urine receives little or nothing; but the vaginal membrane is throughout invested with a lining of pavement epithelium, the elements of which are detached with facility and in great quantity, giving rise to an abundant amorphous-looking, light, cloudy deposit in the urine. When examined microscopically this deposit is found composed of large flat cells, resembling the epithelia of the mouth (Fig. 22). The cells either lie discrete, or united by their borders into patches of rude mosaic.

A deposit of this character is found only in the urine of females, and comparatively few are wholly exempt from it. In the subjects of vaginal leucorrhœa it is always abundant; but it is also present frequently, and in quantity, where there is no appreciable disorder of the genital passages. Even young (female) children may have a sedimentary urine from this cause, especially those of a strumous habit.

The epithelium of the bladder, ureter, and pelvis of the kidney finds its way into the urine of both sexes in cases of vesical calculus, renal calculus, and pyelitis from any cause. The epithelium which lines these parts is of a transitional character, and presents a great variety of forms — cylindrical, spindle-shaped, caudate, oval, spheroidal, and irregular (Fig. 23). It cannot fail to be noticed how like some of these cells are to cancer-cells; so like, indeed, that the recognition of cancer-cells (as such) in the urine becomes a matter of very great uncertainty. In cases of suspected pyelitis the existence of cells of this class in the urine greatly fortifies the diagnosis. (*See* Pyelitis.)

FIG. 23.



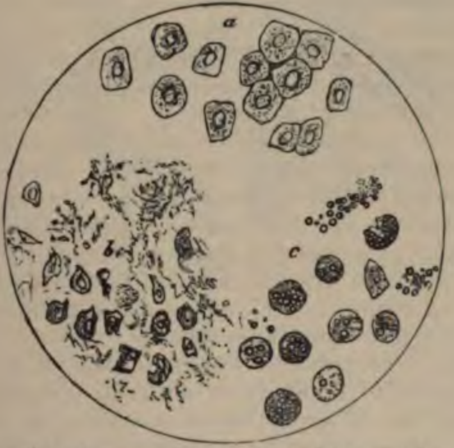
Epithelial cells from the bladder, ureter, and pelvis of the kidney.

II.—RENAL EPITHELIUM AND CASTS OF TUBES; THE DEPOSITS ASSOCIATED WITH ALBUMINURIA.

As renal epithelium and casts of the uriniferous tubes are commonly found together, it will be convenient to consider them in conjunction.

The uriniferous tubes are lined with a single layer of epithelium. The cells of this layer in the cortical part of the kidney consist, in the healthy state, of a round or slightly oval nucleus having a delicate, regular outline, resembling closely, both in size and aspect (except in not being biconcave), the red corpuscle of the blood; around this nucleus is aggregated a quantity of solid, yet friable, faintly granular substance (Fig. 24, *a*). A distinct cell-wall is usually understood to exist around each nucleated mass; but my own observations tend to support the view of Dr. Beale, that, in the convoluted tubes, a distinct cell-wall can only occasionally be seen. When the cut surface of a healthy kidney is scraped, the nucleated masses are freely separated from each other. The nucleus itself is then seen to be exceedingly uniform in size and shape; but the granular matter surrounding it is very irregular. Sometimes the nucleus is quite free; more commonly it is embedded in granular matter. Sometimes this latter forms a spheroidal mass, with a more or less distinct cell-wall; sometimes the granular mass looks as if partly broken off; or there remains only a small quantity of it

FIG. 24.



Renal epithelium. *a*. Natural appearance; *b*. Atrophied and disintegrated renal cells; *c*. Renal cells in a state of fatty degeneration.

adhering to the nucleus. In the straight tubes the cell-wall is always distinct enough, and the cells are flatter, so that the available bore of the tubes is larger in the pyramidal than in the cortical portions. When there is rapid proliferation of the epithelium of the uriniferous tubes (as in the large white or mottled kidney) the nucleus is frequently seen cleft into two or three nucleoli, and the cell puts on the appearance of a pus corpuscle.

The epithelial lining of the uriniferous tubes is liable to be separated from the basement membrane, in certain diseased conditions, and discharged with the urine. Coagulable matter is also liable to be poured into the uriniferous tubes, and having solidified there is afterwards washed out by the stream of urine, and appears therein as casts or moulds of the tubes.

The epithelium and casts thus discharged present a number of modifications of form and aggregation, which serve to indicate and distinguish particular states of disease in the kidneys.

Renal epithelium, forming a urinary deposit, occurs usually in scattered patches: but in the acute form of Bright's disease the epithelium is detached in coherent pieces, which constitute casts of the entire lumen of the tubes. These are "the epithelial casts" to be presently mentioned. The cells never, perhaps, preserve perfectly their normal form. The most common change is a greater or less disintegration or breaking up of the cells into amorphous granular matter, which readily takes place from the absence or extreme tenuity of the cell-wall. The cells also become atrophied (Fig. 24, *b*); and not unfrequently degenerated into fatty corpuscles (*c*), which are significant of changes in the kidney of most serious nature.

CASTS OF THE URINIFEROUS TUBES.—Casts of the uriniferous tubules, composed of a fibrinous material, are often found in the urine, and are of great importance in the semeiology of renal disease. As a rule they are associated with albuminuria, but they are occasionally detected when the urine does not contain any albumen discoverable by our ordinary tests. Tube-casts present the following varieties:

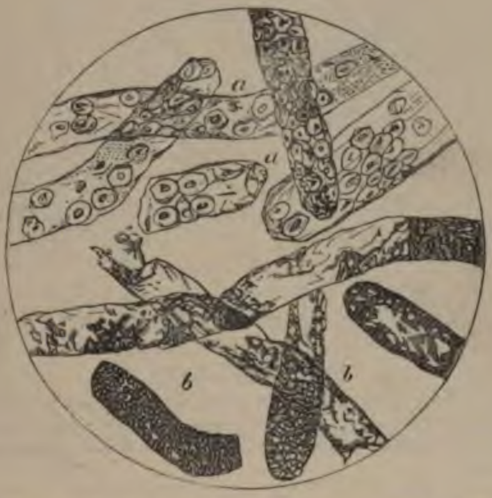
1. *Epithelial Casts* (Fig. 25, *a a*).—These consist of a cylinder of coagulable matter, studded over with epithelial cells which adhere thereto and are partly embedded therein. Some epithelial casts seem to be entirely composed of amalgamated epithelial cells.

2. *Opaque Granular Casts* (Fig. 25, *b b*).—These have a dark coarsely granular appearance, and are generally of medium size ($\frac{1}{700}$ of an inch in diameter).

3. *Transparent or Waxy Casts* (Fig. 26).—These are clear, glassy, fibrinous cylinders, sometimes so transparent as to be invisible until tinted artificially by means of iodine or a solu-

tion of magenta; sometimes faint markings map their surface, or they show a faint molecular composition. They present extreme differences of diameter; the smallest are not more than the breadth of a blood corpuscle (*a a*); the largest are $\frac{1}{500}$ of an inch, or more, in breadth (*b b c*); others again are medium sized.[1]

FIG. 25.



a a. "Epithelial" casts. *b b.* "Opaque granular" casts, from a case of acute Bright's disease.

4. *Fatty Casts* (Fig. 27, *a a*).—Sometimes a transparent cast is studded with tolerable uniformity with minute oil particles; more commonly the oily particles are irregularly distributed in and on such a cast; sometimes again they are collected into dark botryoidal masses — apparently the result of the breaking up of an adherent cell which has undergone fatty degeneration.

5. *Blood Casts* (Fig. 27, *b b*).— Sometimes these are exceedingly beautiful objects, being perfect cylinders composed of delicate circles placed in apposition; more generally a fibrinous cast is studded irregularly with blood-corpuscles, some perfect, and some withered and contorted; sometimes the cast seems composed of blood-disks crushed or compressed into a cylindrical mould (*c*).

6. *Pus Casts.*—Dr. G. Johnson has described and figured moulds or casts of the uriniferous tubes composed of pus corpuscles. In two such cases, examined post mortem, he found

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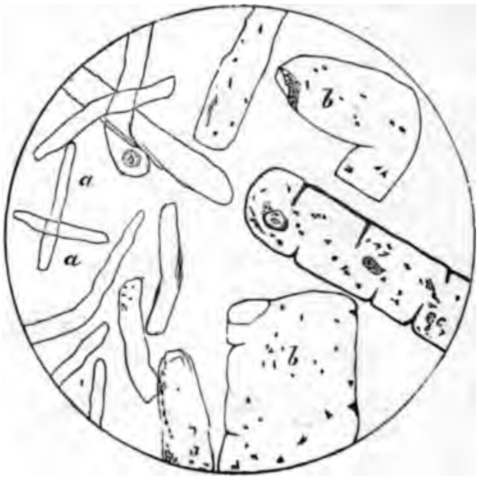
[1] Bartels has described an amyloid change occasionally found in these casts.

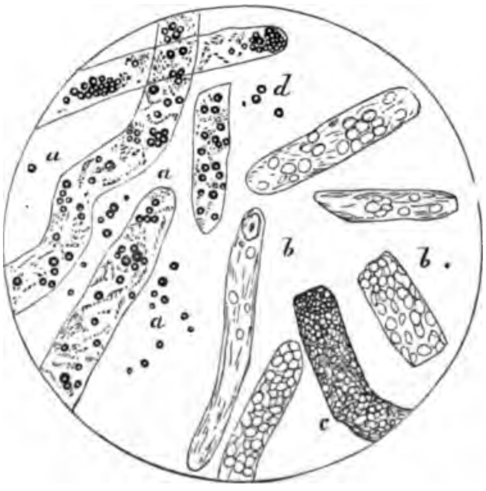
multiple abscesses in the kidneys. In a case examined by myself, where both kidneys were riddled with myriads of secondary abscesses, the urine found in the bladder after death contained no recognizable tube-casts; the observation was,

FIG. 26.



Waxy casts. *a a*. From the urine of a man with chronic Bright's disease of eight months' duration (urine bloody, intensely albuminous, anasarca, dying from pneumonia); *b b*. From a case of chronic Bright's disease (large white kidney); *c*. From a case of chronic Bright's disease (contracted kidney with fatty degeneration).

FIG. 27.



a a. Fatty casts; *b b*. Blood casts; *d d*. Free fatty molecules.

however, an imperfect one, owing to commencing ammoniacal decomposition of the urine, which may have caused disintegration of any preëxisting casts.

The basis material, or substratum composing tube-casts, was supposed by Henle to be coagulated blood-fibrin. This is probably true with regard to blood casts, and the recent researches of Weissgerber and Perls,[1] Posner,[2] and Ribbert[3] tend to support the view that hyaline casts may be formed in this fashion, and that possibly the disintegration of leucocytes may play some part in causing the coagulation. But the investigations of Axel Key and Langhans[4] have made it probable that many hyaline and granular casts are otherwise composed. According to these investigations, most tube-casts are formed by fusion and disintegration of cells which have escaped into the lumen of the uriniferous canals. These cells may be desquamated epithelial cells, leucocytes, or blood-disks. In the canals, the disintegrated material is moulded into cylinders, and may then undergo further changes, becoming transparent or finely granular; some casts are also to be regarded as a secretion product of the epithelial cells lining the tubules. Cornil[5] has observed hyaline globules in the epithelial cells, which are extruded into the lumen of the tube and combine to form casts. In their passage through the lower renal tubes the casts may undergo various alterations in form.

To the naked eye deposits of renal epithelium and tube-casts appear amorphous; they are often very scanty, and resemble a cloud of mucus; sometimes they are more dense and form a white flour-like sediment.

CLINICAL SIGNIFICANCE OF RENAL EPITHELIUM AND TUBE-CASTS.—The most general inference from the presence of these bodies in the urine, is the existence of some serious disorder in the kidneys. But a study of their various forms and appearances furnishes still further information of great weight in the diagnosis and prognosis of the different stages, and different types of renal disease. This subject cannot, however, be advantageously considered in the present section, but will take its place more appropriately in the chapters on Bright's disease and congestion of the kidneys. The following general remarks may, however, find room here:

1. The deposit may, and generally does, contain a mixture of two or more varieties of casts and cells.

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[1] Arch. f. Exper. Pathol., vi. p. 130.

[2] Virch. Archiv., vol. 79.

[3] Centralbl. f. Med. Wissensch., 1879, p. 886.

[4] Virch. Arch., vol. 76.

[5] Journal de l'Anatomie, 1879, p. 402, and Practitioner, 1882. (On the subject of Casts, *see* Ziemssen's Cyclopædia, v. 15, p. 75, and Supplement to the same, p. 639.)

2. Conclusions as to their pathological meaning must be deduced from the prevailing types rather than from the absence or presence of one or two of a particular character. For example, it must not be assumed that the kidneys are in a state of hopeless fatty degeneration, or even commencing to undergo that change, because one or two cells, or one or two casts, display oil molecules.

3. It is necessary, in order to avoid serious errors, to examine specimens of urine passed on two or three separate days.

4. Bearing in mind these precautions, and having regard to the previous history of the case, the following conclusions are *generally* warranted. (*a*) Epithelial casts and blood-casts indicate a disease of recent origin. (*b*) Transparent large waxy casts, mixed with dark granular casts, indicate a chronic disease. (*c*) Epithelium and casts containing much fat indicate fatty degeneration.

TUBE-CASTS WITHOUT APPRECIABLE ALBUMINURIA.—Tube-casts are occasionally found in urinary deposits, when our most delicate tests fail to detect albumen in the urine. In cases of passive renal congestion from emphysema or regurgitant heart disease, small hyaline casts specked with granules are often found without appreciable albuminuria. Similar casts are also found in cases of icterus, as has been pointed out by Nothnagel and Finlayson.[1] In acute Bright's disease, the discharge of tube-casts sometimes continues after the disappearance of the albumen, and in cases of chronic Bright's disease, when the albumen temporarily disappears, casts are still sometimes found in the deposit.

In most of these cases it may probably be assumed that albumen is really present in the urine, but in so small a quantity that it is inappreciable to our ordinary tests. It has been too hastily assumed that our tests for albumen are infinitely delicate, but this is not the case. In urines of low density, where the normal ingredients are in comparatively small proportion, nitric acid is an extremely sensitive test, but when the urine is more concentrated, and the normal ingredients are in larger proportions, a not inconsiderable amount of albumen may be present, and yet fail to give any reaction with nitric acid. A simple experiment is sufficient to prove this. Take an ordinary albuminous urine, and dilute it with water until it gives an opalescence with nitric acid only after the lapse of forty-five seconds after the addition of the test. Suppose that this point is reached when the urine is diluted with 200 times its bulk of water. At this point the diluted urine contains 0.0034 per cent. of albumen.

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[1] *See* an interesting paper, "On Tube-casts without Albuminuria," in the Brit. and For. Med.-Chir. Rev. for January, 1876, by Dr. Finlayson.

If now, instead of diluting with water, we dilute to the same point with a healthy non-albuminous urine, neither nitric acid nor heat will detect the least trace of albumen, though, of course, it must contain the same amount as when it was diluted with simple water.

III.—FATTY MATTER IN URINE.

It appears highly probable, from the investigations of Dr. Schunck, that fatty matter is contained in minute quantities dissolved in normal urine. From 45 litres of healthy urine he obtained 0.14 gramme. The proceeding he adopted was to filter successive portions of urine through purified animal charcoal. The charcoal was then dried and treated with boiling alcohol. By repeated evaporations of alcoholic solutions he obtained a quantity of a white solid crystalline fat which he considered to be a mixture of stearic and palmitic acids.[1]

Pathologically fatty matter appears in urine under a variety of circumstances.

1. In the preceding section it has been stated that tube-casts and renal epithelium (sometimes vaginal epithelium also) are liable to undergo fatty degeneration, and oily particles then appear in the urine, either enclosed in the altered cells or lying free.

2. In the condition called chylous urine, free fat is discharged in great quantity, either in the form of globules visible under the microscope, or, more commonly, divided into molecules so small that they appear only as granular particles, under the highest magnifying powers. (*See* Chylous Urine.)

3. The discharge of quantities of fluid fat by the kidneys is a phenomenon so extraordinary and unexpected, that its occurrence has been doubted. There is no doubt, however, that cats and dogs, fed with an excessive quantity of fat, excrete oily matter in considerable proportion with the urine, so as to yield globules visible with the microscope. There appear to be also a few well-authenticated instances of the occurrence of this phenomenon in the human subject. The late Mr. Turner informed me that such an instance fell under his own notice. The patient was taking cod-liver oil, and each day there was a discharge of yellow oil with the urine. Two examples have also been brought forward by Dr. C. Mettenheimer; one was a man with cancer of the lungs, who was taking daily a tablespoonful of cod-liver oil; the second was a woman convalescent from acute nephritis, who was taking the emulsio-cannabina.[2] Dr. Hen-

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[1] "On a Crystalline Fatty Acid from Human Urine," by E. Schunck. Proc. Roy. Soc., 1867.

[2] C. Mettenheimer, Archiv. d. Verein, f. Wissensch. Arb., Bd. i. p. 374. *See*, also, A. G. Long's Dorpat Thèsis, "De adipe in urina et renibus, etc." (1852), and Rassmann, Thèsis, "Ueber Fettharn," abstracted in Virch. and Hirsch. Jahresb., 1881.

derson likewise describes three cases of heart disease, in which oil globules appeared on two or three occasions in the urine ("Brit. Med. Journ.," 1858). Mr. Bowen ("Brit. Med. Journ.," 1882) relates a case of phthisis, treated by cod-liver oil, in which oil globules appeared in the urine and vanished after the administration of the oil was stopped.

Other cases are reported, by Dr. Oliver, in the "Brit. Med. Journ." of 1879.

4. Concretions containing fatty matter have been encountered in the urinary bladder. (*See* Urostealith.)

5. CHOLESTERINE.—Dr. Beale has shown that the oily particles so frequently seen in the urinary deposits of chronic Bright's disease contain cholesterine in a dissolved state, but spontaneous deposits of cholesterine crystals appear to be of extreme rarity. The following case is the only example which has fallen under my observation:

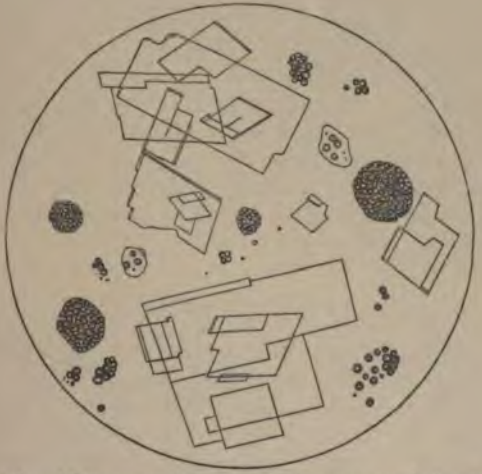
T. M., æt. 31, a pale but not unhealthy looking mechanic, called on me September 28, 1871. He stated that for three years he had been suffering from frequent hæmaturia with pain and aching in the site of the left kidney. The symptoms corresponded closely with those of a case of left renal calculus. Careful and repeated examination of the loins yielded no physical signs; the patient had never voided any stone or palpable gravel, but he often voided, with considerable suffering, cylindrical worm-like bodies, composed of fibrine, which I judged to be casts of the ureter.

At my request he voided urine into a clean glass. The specimen was very bloody. Under the microscope, even before the deposit had settled, I discovered numerous plates of cholesterine. These were mingled with large numbers of the so-called "granular corpuscles," and aggregations of oily particles of various sizes and shapes, together with free fatty molecules (*see* Fig. 28). Blood-corpuscles were, of course, abundant; and these were mixed with transitional epithelial cells—cylindrical and irregular —such as are derived from the pelvis of the kidney; but no pus globules. There were no casts of tubes, and the proportion of albumen did not exceed that which might be accounted for by the blood. The general health was remarkably good, and the general complexion of the case bore no resemblance to any type of Bright's disease. A week later the patient called on me again, and brought a specimen of the urine which he had voided on the morning of the same day. It had a similar appearance to the one just described, but it contained much less blood. It contained large quantities of granular corpuscles and free oily molecules, but only a few cholesterine crystals. He again voided urine in my presence, and in this cholesterine crystals were found in considerable numbers. In other respects it resembled the preceding specimens. This man continued under my observation for several weeks, and the urine invariably exhibited the same objects under the microscope. I was not able to satisfy myself as to the cause of these peculiar appearances. I suspected the existence either of a hydatid cyst opening into the pelvis of the kidney, or commencing general cystic degeneration of the organ; but I could not discover any echinococci hooklets in the urine, nor any physical signs of renal tumor.

When I last saw this man, on January 3, 1872, very marked improvement had taken place under the use of the Liq. ferr. pernit. The blood in the urine had, for six weeks, been reduced to a trace, but there still existed in the deposit numerous crystals of cholesterine, and abundance of the fatty and granular particles.

The only case hitherto recorded at all comparable with the above, so far as I know, is one described by Dr. Murchison. The patient was a man of fifty-four, who, for fourteen years, had passed large quantities of pus with the urine. When he came under observation, three days before his death, the urine was very purulent, and contained large numbers of cholesterine crystals. At the autopsy the right ureter was found blocked up, but not entirely, by a calculus. The kidney was wholly converted into a large (pyonephrotic) cyst, containing pus rich in cholesterine crystals. The left kidney was also converted in the same way, but not so completely, into a suppurating sac through the blocking up of its pelvis with a large branching stone. The pus in the left kidney did not contain cholesterine crystals.[1]

Fig. 28.



Cholesterine crystals and fatty aggregations and molecules spontaneously deposited in the urine in the case of T. M.

Reference may be made here to a curious case, reported by

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[1] Path. Soc. Trans., xix. 278. Pöhl (Virch. and Hirsch. Jahresbericht, vol. i., 1877) found cholesterine in the urine of an epileptic, but the observation was superficial.

Ebstein,[1] of pyonephrosis with a tumor in the flank in which fat globules were found in the urine, together with crystals of hæmatoidin.

6. KIESTEINE.—This is a name given by Nauche to a peculiar pellicle said to form on the urine of pregnant women when left at rest for a few days, and said to contain fatty and caseous matter. Much has been written on the nature of this pellicle and its value as a sign of pregnancy; but the accounts are so contradictory that no safe conclusions can be drawn from them. I have carefully looked over all the observations hitherto made on the subject, and am inclined to believe that the kiesteine pellicle is nothing more nor less than the mould fungus, which is apt to grow luxuriantly in urines containing organic matters. The urines of pregnant women are likely to form a fitting nidus for this fungus from the large quantity of epithelial débris which they generally contain. A very full account of the literature of this subject is given in Montgomery's "Signs and Symptoms of Pregnancy." A paper, by R. C. Golding, in the "British Obstetric Record" for 1847–48, and another, by Hicks, in the "Lancet" for 1859, vol. ii. p. 281, may also be consulted. The question deserves to be reëxamined; but the investigation, to be of use, must be conducted with much more rigorous exactness than any hitherto published.

IV.—PUS IN URINE.

Urine containing pus is turbid and milky when voided. After standing a while, it deposits a dense yellowish-white sediment. Pus presents a very different appearance, according as the reaction of the urine is acid or alkaline. In the former case the deposit is loose, and the corpuscles discrete; but if the urine be alkaline, as it often is, from ammoniacal decomposition, the pus coheres into a viscid tenacious mass, which can be drawn out into long tough strings. The latter appearance is diagnostic of pus.

MICRO-CHEMICAL CHARACTERS.—Pus possesses an analogous constitution to blood, and is composed of cellular particles floating in a liquor puris. Liquor puris, like liquor sanguinis, is an albuminous saline fluid; therefore purulent urine necessarily contains more or less albumen—the quantity varying, according to the proportion of pus present, from a trace too slight for detection by ordinary reagents, to a considerable impregnation. It is sometimes a point of importance, and always of considerable nicety, to decide whether the quantity of albumen in a purulent urine is no more than can be accounted for by the pus present, or whether some of it is not due to renal disease. Such a question occasionally arises in cases of vesical calculi accompanied with catarrh of the bladder—most surgeons holding that the coexistence of renal degeneration constitutes a bar to operation. Usually, purulent urines become merely hazy with nitric acid; and the quantity of pus must be very great, indeed, to account for a large deposit of albumen. Important assistance in doubtful cases is to be obtained by a diligent search for tube-casts in the freshly voided urine.

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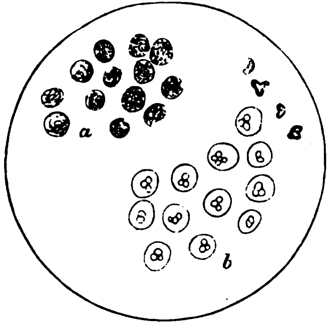
[1] Deutsch. Arch. f. klin. Medic., vol. xxiii.

FIG. 29.



Pus globules. *a*. Without reagents; *b*. After the addition of acetic acid.

The chemical test for pus is the conversion of it into a viscid mass by the addition of Liq. potassæ or Liq. ammoniæ.

The pus corpuscle is a spherical cell, about one-third larger than a red blood-disk. Examined without reagents, it appears opaque, granular on the surface, and yellowish (Fig. 29, *a*). The denser the urine the smaller and more crumpled becomes the pus corpuscle; whereas, the addition of water expands and clears it—sometimes bringing into view the nucleus. This effect is brought about much more quickly and powerfully by a drop of acetic acid, insinuated beneath the covering-glass. The nucleus thus displayed is found to be cleft into two, three, and sometimes four nucleoli (*b*). If the acid be added in excess, the cell-wall and contents disappear altogether, and the cleft nuclei float free in the fluid.

CLINICAL SIGNIFICANCE.—The importance of pus in urine depends on its source and quantity. Suppuratiom may take place in any part of the genito-urinary passages, or abscesses of adjacent parts may burst into these and cause pus to appear in the urine. It is therefore always desirable to decide the anatomical source of the pus. This is not always easy, and sometimes it is impossible. The following are the points to be held in view in such an inquiry:

When pus is derived from the urethra (as in gonorrhœa) a drop or two may be squeezed from the meatus urinarius by compressing the penis. Gonorrhœa is the commonest cause of pus in the urine of men. The quantity is always small and the general properties of the urine are not affected thereby.

In women the most common cause of slight admixtures of pus with the urine is leucorrhœa, which betrays itself by the coexistence of abundance of pavement epithelium.

Pus from the bladder has a more serious significance, as indicating the existence of cystitis. Usually there is little difficulty in tracing this to its right source by the local symptoms. In severe cases, the excessively frequent micturition, the ammoniacal state of the urine when voided, and the speedy gelatinization of the pus into a viscid mass, leave no doubt on the mind of the practitioner. But when the cystitis is slight, and of old standing, there is more difficulty, as the urine may retain its acidity, and micturition may not be very frequent. The presence of stone in the bladder, an enlarged prostate, the history of a past lithotomy, or of an old stricture, generally gives a key to the source of the pus.

Suppuration in the pelvis of the kidney (pyelitis) is generally indicated by direct signs of irritation in the loins. When these are absent, reliance must be placed on finding with the pus, epithelial elements of transitional character (*see* Fig. 23), an acid reaction of the urine, and absence of signs pointing to the bladder and urethra.

The bursting of an abscess into the urinary passage is usually signalized by the sudden irruption of a large quantity of pus into the urine. Perineal abscesses opening into the urethra can scarcely be overlooked; but perivesical and perirenal abscesses are more difficult to diagnosticate.

Purulent urine, from suppuration in the kidney, will come under consideration in future pages. (*See* Suppuration in the Kidneys, Pyonephrosis.)

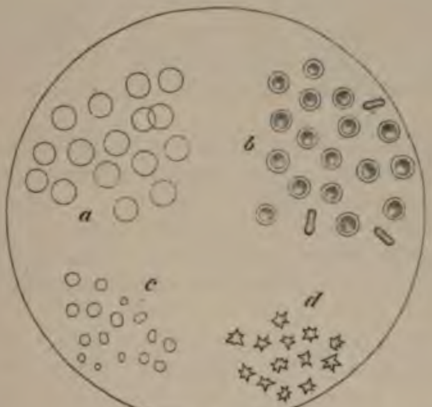
V.—BLOOD IN URINE—HÆMATURIA.

An admixture of blood with the urine is readily recognized by the color which it imparts to the secretion, unless the quantity be very small. If the blood is derived from the kidneys, it is diffused equally through the urine, communicating to it a reddish or a peculiar smoky tint, and after standing awhile a chocolate-colored grumous deposit subsides. But when the blood is derived from some part of the urinary tract below the kidneys—ureters, bladder, or urethra—the color imparted to the urine is pinkish or vermilion, and frequently distinct clots are found in the deposit.

The microscope is the surest means of discovering blood in urine; nevertheless, the corpuscles may disappear very speedily if the urine be of very low specific gravity, or ammoniacal. In acid urine of moderate density (1020–25) the corpuscles remain visible and preserve their form for several days.

They do not run into rouleaux in the urine, as they do when drawn directly from the bloodvessels, but stand discrete and separate. In dilute urine the corpuscles expand somewhat from imbibition, and appear under the microscope as pale circles with sharp delicate outlines, and without any appearance of cell-contents (*see* Fig. 30, *a*). If the urine be more concentrated, they preserve more nearly their normal biconcave contour, and appear smaller and more deeply shaded (*b*). Sometimes they shrink and crumple and become misshapen in various ways (*c d*).

FIG. 30.



Blood-corpuscles in urine. *a.* Slightly distended by imbibition; *b.* Showing their biconcave contour; *c.* Shrivelled; *d.* Serrated.

The marks by which blood-corpuscles are distinguished from other cells found in urine are, the extreme tenuity of their outline, the absence of visible cell-contents, and especially of a nucleus, and their feeble refractive power. When the biconcave form is preserved, this, of course, is diagnostic. Blood-disks are liable to be confounded with confervoid sporules, with the minute discoid forms of oxalate of lime, and with the nuclei of renal epithelium. From the first, they are distinguished by the absence of a nucleus, which can, with a good glass, always be detected in the sporules. Sporules also generally are somewhat oval, often elongated, and show signs of budding. The discoid crystals of oxalate of lime are distinguished by the existence of intermediate forms which connect them with dumb-bells. Renal nuclei are distinguished by their strong refraction, by being strongly tinted by magenta,[1] and usually they are surrounded by some portion of the material which originally invested them. The coloring matter of the blood in urine may also be detected by the spectroscope or by the guaiacum test. The latter is conveniently carried out in the following manner: Pour a little urine into a narrow test-tube and add to it one or two drops of tincture of guaiacum and a little ozonic ether. Shake well and allow to stand for a few moments. If blood-coloring matter is present, the ether on rising to the top of the mixture will be colored blue.[2]

Urine containing blood is of necessity always more or less albuminous. The quantity may be so great that the urine looks like pure blood, and coagulates spontaneously, or so small that the microscope is required to detect it. The hemorrhage may arise from a great variety of causes, which may be classified as follows:

1. *Local lesions*—external injury, violent exercise, calculous concretions, ulcers, abscesses, cancer, tubercle, parasites, active or passive congestion, Bright's disease.

2. *Symptomatic*—in purpura, scurvy, eruptive and continued fevers, intermittent fever, cholera, etc., mental emotion.

3. *Supplementary or vicarious*—to menstruation, hemorrhoids, asthma.

Cases also occur which are not referrible to any of these categories, of which the origin is extremely obscure.[3]

1. HÆMATURIA FROM LOCAL LESIONS.—This division includes by far the largest number of cases. A point of great importance is to ascertain the exact source of the blood. This is not, as a rule, difficult.

Hemorrhage from the *substance of the kidney* is recognized by the existence of tube-casts in the deposit. By far the most common cause of this variety of hæmaturia is some form of Bright's disease or its allies (congestion, etc.). In falls and blows on the loins, or any injuries supposed to implicate the kidneys, the occurrence of casts in the urine furnishes a valuable diagnostic sign. In the following remarkable case of laceration of the kidney from a fall, the condition of the urine was accurately noted from the time of the accident till death.

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[1] *See* a paper by the author, "On the Effects of Magenta and Tannin on the Blood-corpuscles," in the Proceedings of the Royal Society for 1863.

[2] If the patient is taking iodide of potassium, or if saliva is mixed with the urine, the test is inapplicable.

Heller's test for blood in the urine consists in boiling the urine with liq. potassæ. The precipitate of phosphates which is then thrown down is colored brown if blood be present.

[3] It may be necessary to remind students that in females the urine is generally bloody during the menstrual flow; it may also become so at any time if there be uterine and vaginal hemorrhage.

E. Davis, a bricklayer, aged 36, was brought into the Manchester Infirmary at 3 P. M. on April 27, 1863, in a state of complete insensibility, with gasping respiration, apparently dying. In the course of two hours he recovered consciousness, and answered questions imperfectly, in a half-drunken manner. It appeared that he went to his work in the afternoon intoxicated, and that he had fallen a height of seven stories. There was a compound fracture of the skull, and the legs were severely contused and lacerated. From the time that he recovered speech the patient continued to talk in a curiously incoherent manner, as if he were drunk—except that the pronunciation of words was unaffected.

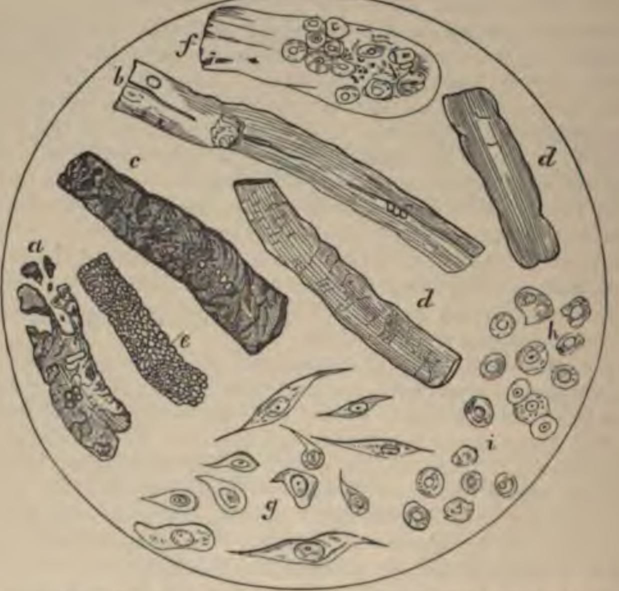
No urine was passed on the day of the accident; but on the day following about eight ounces were withdrawn by catheter. The urine was excessively bloody, dark chocolate-colored, and highly albuminous.

On the third day (April 29) the patient was in the same state. No urine was passed spontaneously; at 8 P. M. about an ounce was withdrawn by catheter; it was of the same character as before, but less bloody, and less albuminous. On the morning of the fourth day I found the patient breathing rapidly, with a quick small pulse; the tongue was moist; there was great thirst—no appetite; the bowels had been opened several times by medicine. At 9 P. M. of the same day I again visited the ward. No urine had been voided, and the bladder was not distended. The general condition was evidently worse; the delirium was constant, and he swore awfully when his legs were touched.

At noon on the fifth day the patient was much weaker; the breathing was interrupted; he muttered incoherencies unceasingly; and waved his hands as if he saw spectres in the air; he picked and tore the bedclothes; he had torn three sheets to ribbons, and had torn the counterpane. He did this quietly, without violence, and without attempting to get out of bed. When asked questions he answered quite at random; the tongue was dry and red; pulse almost imperceptible. No urine had been passed spontaneously this day, nor the day before. The house surgeon introduced a catheter, and succeeded, by compressing the abdomen, in withdrawing about two ounces of a yellowish urine, with small, dark, chocolate-colored granules floating in it. About an hour after, the patient died quietly, without coma or convulsions.

During the five days that the patient survived no urine was passed spontaneously; but eleven ounces were withdrawn by catheter at three different times. The first specimen, drawn the day after the accident, was excessively bloody; the second, drawn on the third day, was much less bloody; the third, drawn just before death, contained no liquid blood, and had a yellow color, but it deposited a considerable sediment of chocolate-colored granules which consisted of indurated clots of blood. Although this last specimen, consisting of only two ounces, was the product of forty hours' secretion, its specific gravity was only 1015, and its proportion of albumen only $\frac{1}{12}$. The microscopic examination of the deposits revealed the existence of an immense quantity of casts of the uriniferous tubes, and these changed character as time passed over. In the first specimen the casts were all dark, opaque, and granular (Fig. 31, *a*, *c*, *e*), evidently composed of crushed blood-clot; no free renal epithelium, nor any pyelitic cells, were found. In the second specimen, in addition to the dark granular casts, there were numerous deep-brown plain casts, with strongly marked outlines and very few markings (*d*, *d*); a few transparent casts were also found, some of them studded with epithelium. In the second and third specimens free renal epithelium (*h*, *i*), and epithelium from the pelvis and infundibula (*g*), appeared in great abundance. The renal epithelium was deeply browned, evidently from hæmatine, but was otherwise natural. Many of the casts had dumb-bells embedded in them.

FIG. 31.



a, *c*, *e*. Dark granular casts; *b*, *d*. Yellow plain casts; *f*. Large transparent cast studded with epithelium; *i*, *h*. Free renal epithelium, *i*, before, and *h*, after the addition of acetic acid; *g*. Cells from the pelvis and infundibula.

Autopsy forty-eight hours after death. Left parietal bone fractured, with a slight depression. Dura mater not lacerated; no free blood on or under the membrane; but there was an ecchymotic patch on it as large as a florin, corresponding to the fracture. There was no blood in the arachnoid space; but the pia mater was injected over the space of two square inches in the vicinity of the fracture. No lymph was thrown out on any part, nor was there softening or other abnormal condition of any portion of the brain.

Abdomen. There was no external sign of direct violence over the loins; all the abdominal organs, except the kidneys, were uninjured and healthy. *Left kidney* weighed 9¼ oz.; it was not lacerated. On section minute granules of indurated blood were found in several of the infundibula; the whole gland was hyperæmic. *Right kidney* weighed 9½ oz., was torn in two places on its posterior aspect. The lacerations ran across, somewhat crookedly, from the outer border almost to the hilum; they were about an inch apart, and varied in depth from one to three or even four lines. They were completely closed by a wedge-shaped solid clot of blood, which was very firm, and, where in contact with the renal substance, bleached. The renal tissue immediately adjacent to the lacerations appeared perfectly natural—neither injected nor softened. The tunica propria was, of course, torn through over the site of the lacerations. The lacerations did not penetrate in any part to the infundibula, but two large, firm, blood-concretions—one as large as a horse-bean, and the other as large as a pea—lay loose in the pelvis, and several smaller ones were found in the infundibula. The perirenal adipose tissue was deeply stained with blood on both sides; but it contained neither fluid blood nor clots. The peritoneum was not injured nor inflamed. The *heart* and *lungs* were healthy.

It was evident that the direct cause of death in this case was suppression of urine—aided, perhaps, by a degree of delirium tremens. The reason, probably, why no signs of inflammation were found in the brain and peritoneum was, that the patient never really rallied from the shock of the accident; and that reaction never properly took place. The desquamation of the epithelium of the pelvis and infundibula must be attributed to the irritation of the blood-concretions found therein.

Hæmaturia is rarely serious from its quantity in any form of Bright's Disease, and is generally quite insignificant. Far more serious are the consequences of the coagulation of the effused blood in the uriniferous canals. Unless these plugs are expelled by the pressure of the urine from behind, they permanently block up the tubes and destroy the function of the corresponding portions of the gland. Hence, any hemorrhage from the substance of the kidney, however it may arise, is attended with serious hazard that the foundations of a fatal renal degeneration may be laid thereby.

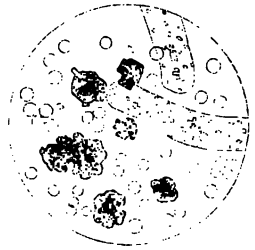
Cancer of the kidney is often associated with profuse and repeated hæmaturia; the diagnosis rests chiefly on the presence of a tumor in the loins. (*See* Cancer of the Kidney.)

The endemic hæmaturia of Mauritius, Brazils, Cape of Good Hope, Egypt, and some other hot countries, which so greatly puzzled pathologists in times past, seems to have found its explanation in the presence of a minute parasite which infests the mucous membrane of the pelvis of the kidney and the bladder. The researches of Griesinger, Bilharz, and Dr. John Harley on this subject will be described in the chapter devoted to parasites of the kidney. (*See* Bilharzia Hæmatobia.)

In tubercle, abscess, renal embolism, hydatids, the hemorrhage is seldom more than trifling. In active congestion of the kidneys after taking turpentine or cantharides, the bleeding is sometimes severe. As these classes of cases are treated separately in subsequent parts of this work, it will not be necessary here to go into further details.

Sometimes minute calculous concretions are formed within the tubuli uriniferi, and occasion hæmaturia, which is apt to recur again and again. In these cases crystals or microscopic calculi of uric acid, or oxalate of lime, may often be discovered by a careful examination of the urinary deposit (*see* Figs. 32 and 33). Hæmaturia from this cause may be unaccompanied by any pain beyond a slight aching or sense of fatigue in the loins. Transparent fibrinous casts are also visible in these cases, speckled all over with crystalline molecules, and more albumen is present in the urine than corresponds with the amount of blood voided.

Fig. 32



Microscopic calculi of uric acid, with fibrinous casts dotted with crystalline molecules of uric acid in a case of recurrent hæmaturia.

Hemorrhage from the *pelvis of the kidney and ureters* is commonly due to calculous concretions; much more rarely to cancer, tubercle, cystic disease, or parasites. When the blood has this source the diagnosis turns on the existence of symptoms of pyelitis, nephritic colic, and the passage of a foreign body down the ureter. Sometimes the blood coagulates in the ureter, and long vermicular clots may be afterwards recognized in the urine. The passage of these clots along the ureter produces precisely the same symptoms as a calculus passing in the same direction.

Hemorrhage from the *bladder* is usually recognized by symptoms pointing directly to that organ, namely, excessively frequent micturition, pain in the hypogastrium, and at the neck of the bladder, etc. Exploration of the bladder will generally reveal the existence of calculi or fungoid growth. Varicose enlargement of the veins of the mucous membrane and acute cystitis are also occasional causes of vesical hemorrhage.

Urethral hemorrhage is known by the escape of blood in the intervals of micturition.

Symptomatic Hæmaturia.—Purpura hemorrhagica is occasionally marked by severe hæmaturia. In a case under my care some years ago, there occurred first violent epistaxis requiring plugging of the nares; then profuse hæmaturia set in; when this subsided, the patient rapidly succumbed to intracranial hemorrhage. Scurvy is more rarely attended with hæmaturia. The eruptive and continued fevers, cholera, and yellow fever, are sometimes the occasion of hæmaturia, which is generally a very unfavorable symptom.

SUPPLEMENTARY HÆMATURIA.—Many curious examples have been recorded in which hæmaturia appeared to be *supplementary* to some natural function or some diseased condition. Chopart[1] relates a case in which hæmaturia supplemented a hemorrhoidal flux; Latour[2] adds another. The latter mentions a singular case of spasmodic asthma, of such severity and persistence that the patient had not been able to lie in bed for eighteen months, which disappeared suddenly on the occurrence of hæmaturia. Chopart and P. Frank relate examples in which the menstrual flux was deviated to the urinary passages, and appeared under the form of a periodical hæmaturia.[3]

Mental emotion seems capable in very rare instances of producing hæmaturia. Basham[4] tells of a shoemaker who was subject to attacks of hæmaturia which always recurred on the occasion of his drunken wife's misconduct. Rayer records an instance in which hæmaturia followed a fit of passion.

TREATMENT.—As hæmaturia is merely a symptom, and a symptom which attends a great variety of pathological conditions, the treatment of the cases in which it occurs is necessarily diverse. Sometimes, however, we are called on to treat hæmaturia for itself—in some cases because of our inability to fathom its exciting cause, in others because the loss of blood is so great that it becomes an urgent object to check it, even though the primary disease of which it is a symptom be irremovable.

In the hyperæmia of the kidneys which occurs in acute Bright's disease, after overdoses of turpentine and cantharides, after blows, falls, muscular efforts, and other external injuries, hæmaturia is a positive relief to the loaded vessels, and were it not that the effused blood is prone to coagulate in the uriniferous tubes, and produce a physical obstacle to the excretion of urine of a most dangerous character, the hemorrhage (unless excessive) might safely be left to its own course. To relieve the congestion in these cases, derivation by the loins (cupping etc.), by the cutaneous surface (baths, diaphoretics), and by the intestines (hydragogue cathartics), must be energetically practised.

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[1] Traité des Malad. des voies urinaires. Segalas's edition, p. 283.

[2] Cited by Rayer, t. ii. p. 25.

[3] Chopart (l. c. p. 282) cites one instance, and Rayer two instances, in which hæmaturia occurred at regular monthly periods in males. One of these was a butcher of Sedan. The circumstance became known, and such was the disgust caused thereby that no one would purchase meat from him.

[4] Basham on Dropsy, 3d ed , p. 312.

When hæmaturia is supplementary to hemorrhoidal discharges, leeches may be applied about the anus. It should be remembered, however, that if the blood be shed from the mucous membrane of the bladder, and not from the substance of the kidney, such a discharge is not to be looked on unfavorably, nor to be rashly suppressed. When moderate hæmaturia occurs vicariously with menstruation, it is to be suppressed only on condition that the normal flux be reëstablished.

Passive hæmaturia *in the course* of zymotic diseases should be carefully distinguished from acute Bright's disease, which sometimes forms a *sequela* to these. In the former, the bleeding is probably from the whole or greater portion of the urinary tract, and not solely, if at all, from the kidneys. The internal remedies of most avail in passive hæmaturia, are the mineral acids, especially sulphuric acid, freely administered.

When our object is simply to treat the hæmaturia for itself—to stay the loss of blood—the first point is to enforce perfect rest, and to apply cold in the most effective manner to the bleeding part. If the kidneys be the source of the blood, ice-poultices should be applied to the loins; if the bladder, iced-water injections may be practised into the bladder, and iced-cloths applied to the perineum and epigastrium. The medicinal hæmostatics which have been found of most service, are gallic acid, acetate of lead, alum, ergot of rye,[1] tincture of muriate of iron, turpentine, and matico. Dr. Golding Bird speaks highly of acetate of lead given frequently and in large doses for short periods. He recommends 3 or 4 grains, with one-fourth of a grain of opium, in a pill every two hours, until six or eight doses have been administered—care being taken to keep the bowels open by saline purgatives. Dr. Prout observes: "When the bladder becomes distended with blood, and complete retention of urine in consequence takes place, recourse must be had to a large-eyed catheter and an exhausting syringe, by the aid of which, and the occasional injection of cold water, the coagula may be broken up, and removed. If the hemorrhage be so profuse that the bladder becomes again distended with blood in a very short time, the injection of cold water into the rectum or bladder is sometimes of great use; and should these means fail, from 20 to 40 grains of alum may be dissolved in each pint of water injected into the bladder, a remedy that seldom fails to check the bleeding even when the cause is malignant disease. I have never known any unpleasant consequences follow the use of this expedient; and have seen it immediately arrest the most formidable hemorrhage when all other means had failed, and when the bladder had repeatedly become again distented with blood almost immediately after its removal."[1]

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[1] The subcutaneous injection of five grains of ergotin seems worthy of trial, judging by the successful results of this method in pulmonary and uterine hemorrhages. See Dr. Ritchie, Practitioner, 1871.

VI.—HÆMOGLOBINURIA—PAROXYSMAL HÆMOGLOBINURIA.

HÆMATINURIA.—Attention has been called by Vogel,[2] Oppolzer,[3] and Mettenheimer,[4] to the escape of the coloring matter of the blood (hæmoglobin) with the urine, unaccompanied by rupture of the capillaries and the presence of blood-corpuscles. The urine in such cases assumes a deep red or blackish-red color, very much as if it contained blood; but no blood-disks can be found under the microscope, nor any fibrin. This condition is invariably accompanied by the presence of albumen in the urine. It is caused by the rapid destruction of the blood-disks in the bloodvessels, such as occurs in that state which is known as "a dissolved state of the blood," in septic, pyæmic, and putrid fevers, and in some extreme cases of scurvy and purpura. In such cases hæmoglobin is set free by the disintegration of the red disks, and appears in the urine. Vogel found that inhalation of arseniuretted hydrogen produced an intense (but temporary) degree of hæmoglobinuria. He produced the same condition artificially in animals by inhalation of the same gas and of carbonic acid; also by the injection of substances into the veins which are known to dissolve and break up the red disks.

Ponfick (Virch. "Arch.," B. 72, S. 273) has described hæmoglobinuria as occurring after the transfusion into an animal of blood from an animal of a different species. He believed that the hæmoglobin of the transfused blood-corpuscles was dissolved in the serum of the recipient animal.

Hæmoglobinuria has also been observed after severe burns (1), in typhoid fever (2), and scarlet fever (3), after fat embolism (4), and in poisoning by hydrochloric acid (5), sulphuric acid (6), pyrogallic acid (7), carbolic acid (8), and chlorate of potash (9).*

A case of hæmoglobinuria from chlorate of potash poisoning, in which death occurred, was described by Dr. Dreschfeld and

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[1] Prout: Stomach and Renal Diseases, 5th edit., p. 421.

[2] J. Vogel: Krankh. der Harnbereitenden Organe, in Virchow's Handbuch der Speciellen Path. u. Therap., Band vi., 2te Abth. p. 539.

[3] Wiener med. Wochensch., 1860, Nos. 25 and 26.

[4] Würzburger Med. Zeitsch. 1862, p. 1.

* (1) *See* Lichtheim, Volckmann's Samml. klin. Vorträg., 134. (2) Immermann, Deutsch. Arch. f. klin. Med., Bd. 12, S. 502. (3) Heubner, *ibid.*, Bd. 23, S. 282. (4) Scriba, Deutsch. Zeitschr. f. Chirurg., 1879, Bd. 12, 118; Riedel, *ibid.*, Bd. 12, S. 118, 1880. (5) Naunyn, Dubois Arch., 1868, S. 413 (6) Bamberger, Centralbl. f. Med. Wissen., 1874. p. 571. (7) Neisser, Zeitschr. f. klin. Medic., Bd. 1, S. 88, 1880. (8) Zur Nieden, Berl. klin. Wochensch., 1881, p. 705. (9) Jacobi, New York Med. Record, 1879, xv., No. 11; Marchand, Virch. Arch., 77, p. 455. Catarrhal jaundice may occasionally be accompanied by hæmoglobinuria. This was exemplified by a case I saw a short time ago in consultation with my colleague Dr. Dreschfeld.

Mr. Stocks, at the International Medical Congress, London, 1881. The patient, a woman, had taken an uncertain quantity of the drug, to relieve a sore throat. Death was preceded by great cyanosis, and the passage of quantities of hæmoglobin from the rectum and vagina, and in the urine.

The clinical significance of hæmoglobinuria depends entirely on the pathological state which occasions it.

PAROXYSMAL HÆMOGLOBINURIA.

(*Synonym—Intermittent Hæmatinuria.*)

Attention was first called in this country to this curious disorder by Dr. George Harley, who published two cases in the "Med.-Chir. Trans." for 1865.[1] Subsequently cases were recorded by Dickinson, Greenhow, Gull, and others; and from an analysis of these, and of personal observations, together with cases reported by Continental observers, the following account has been drawn up:[2]

The disorder is essentially intermitting or paroxysmal in its nature. Each paroxysm begins with a feeling of cold or shivering, resembling the cold fit of ague, and terminates with the discharge of a very dark bloody-looking urine. The symptoms then subside, and the urine at the next micturition, or the one after, is found to have resumed its natural healthy appearance. The recurrence of the paroxysms in different cases is most irregular. In some cases the paroxysm recurs once a day, or even twice and thrice a day. More commonly it recurs on alternate days, or twice a week, or once in ten days, or quite irregularly. The paroxysms are sometimes followed by a hot or sweating stage. The onset of a paroxysm is usually sudden. The patient first experiences coldness of the extremities, followed by general chilliness, which in most cases passes into distinct rigors, accompanied by a feeling of malaise, a disposition to stretch himself, and to yawn. In most cases, a sense of weight, or a dull heavy pain is felt in the loins, sometimes extending to the umbilicus, or passing down the thighs, occasionally there has been noted tenderness over the region of the kidneys; and there is frequently pain, or a feeling of stiffness or weakness, in the lower extremities. Retraction of the testicles has been noted in several cases. Retching is a not infrequent symptom, and vomiting was a prominent feature in a few cases, while the patient sometimes complains of thirst, headache, and drowsiness.

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[1] Dr. Wickham Legg, in his admirable paper on the subject, has directed attention to the fact that Dessler, in 1854, published a complete account of the disorder in Virchow's Archiv for that year.

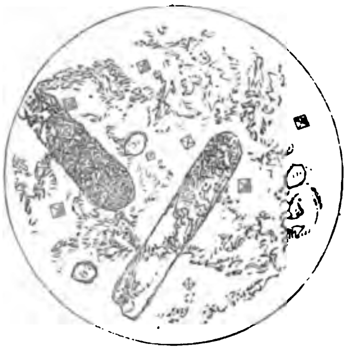
[2] The article on this subject in the preceding edition of this work was the result of an analysis of twenty cases. Subsequently more numerous observations, however, have rendered necessary many alterations and additions.

After these symptoms have lasted for a period varying from thirty minutes to two hours, the patient passes a quantity of dark-colored urine; the pain and general disturbance then subside, leaving the patient apparently quite well till the next paroxysm.

The appearance of the dark urine resembles that of porter or of the darkest port wine. It is generally turbid, and deposits, on standing, an abundant chocolate-colored sediment. The sp. gr. varies from 1015 to 1033, usually ranging from 1022 to 1025. The reaction is either acid or faintly alkaline. It is always highly albuminous, and, on boiling, the albumen coagulates into brownish masses, which, on subsiding, leave the clear supernatant urine of nearly its original dark-red color. In Harley's cases, and in one of mine, the percentage of urea was found to be in considerable excess; in some cases, however, it has been found lower than usual.

The chocolate-colored sediment consists chiefly of amorphous granular matter, which is, presumably, disintegrated blood-corpuscles. Gull found in it myriads of minute crystals of hæmatine. Casts of tubes are also present; these are mostly of dark granular appearance—mixed, however, with a few transparent fibrinous cylinders. Many casts are formed of masses of hæmoglobin. Crystals of oxalate of lime are generally seen, and, very rarely, a few stray blood-disks.

FIG. 33.



Granular matter, casts, and octahedra, from the deposit in the urine of a man with paroxysmal hæmoglobinuria.

The urine gives the usual reactions of blood with the guaiacum and Heller's tests. Usually, on spectroscopic examination, there are found the two absorption bands between Frauenhofer's D and E lines, which are characteristic of oxhæmoglobin. An additional band in the red, indicating the presence of methæmoglobin, has also been observed by many writers. Concerning this band, however, there is some difference of opinion, and no doubt in many cases it is absent.

In most cases the albumen and the hæmoglobin appear and disappear together. Murri[1] and Rosenbach[2] observed albumen in the urine before the blood-coloring matter could be perceived. This, however, has not been confirmed by others. Dr. Forrest, on the other hand, found that the albumen persisted after the disappearance of the hæmoglobin, while Dr. Saundby, in one of his cases, saw the blood-coloring matter in the urine when no trace of albumen could be detected.

During the attack, the extremities, nose, and ears may be cold and cyanotic. A general eruption of urticaria has been noticed in some cases by Mackenzie, Forrest, Lichtheim, and others. In a few cases the liver and spleen increased in size during the attack; and in Boas's case, pains in the region of the liver seemed to be precursors of an attack.

Careful examination of the blood during the attacks has been made by recent observers, particularly by Ehrlich, Boas, and Wolff. The main changes observed were, absence of the ordinary rouleaux of red blood-corpuscles, variations in shape of the red corpuscles, and the presence of Ponfick's so-called "phantom" corpuscles, or red corpuscles from which the coloring matter had been dissolved out. Sometimes, however, the microscopical examinations of the blood have shown no change. The temperature during the paroxysm is usually raised, in one of Dr. Saundby's cases to as high as 105.2° It may, however, be normal, or in some cases a preliminary fall may precede the rise. (*See* Charteris, "Lancet," Jan. 1879.)

The first onset of the disorder is invariably sudden, and can usually be traced to some distinct exposure to cold or wet. The subsequent paroxysms are generally quite unconnected with any fresh exposure to cold, but in other cases the contrary is the case. The paroxysms recur, in some cases, with the regularity of real ague, for weeks together; in other cases the periodicity is quite imperfect. Each paroxysm lasts from three to twelve hours, and it is noteworthy that no paroxysms occur at night, the urine voided before breakfast being invariably natural.[3] The change in the characters of the urine may take place with the utmost abruptness; that passed at one micturition being porter-like, and at the next straw-colored; or it may more gradually become pale, resuming its normal appearance at the fourth or fifth micturition after the paroxysm.

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[1] Revista clinica di Bologna, 1879.

[2] Berlin. klin. Wochen., 1880, p. 132.

[3] *See*, however, a case reported by Lépine (Révue Mensuelle, 1880, p. 722), in which cold had no influence, and the paroxysms occurred at midnight.

The state of the general health seems to vary somewhat. In one case reported by Dr. Dickinson, the patient had the appearance of robust health the day before an attack came on. In most cases, however, the patient has presented a somewhat sallow and icteric aspect, or has looked anæmic, pale, and sickly. Sometimes there has been hepatic derangement distinctly present at the time the patient has come under observation. In one case, an intercurrent attack of jaundice came on, the urine being deeply colored with bile, and copiously depositing lithates, but containing no blood-coloring matter, and only once a trace of albumen. Occasionally, after severe paroxysms, the conjunctivæ are of a yellowish color, but no bile pigment is found in the urine. The color is probably due to serum holding hæmoglobin in solution, which is transuded during the attack.

A rheumatic tendency is a frequent concomitant of this affection, many of the patients having frequently suffered from rheumatism in various forms. In one of my own cases the patient began to suffer from subacute rheumatism, with swelling and pain in the joints after the cessation of the paroxysms. A bronchitic and asthmatic tendency has also been observed in one or two instances. Fraentzel[1] is of opinion that there is a decided tendency to chronic lung disease.

Of twenty cases collated by me, four had at one time or another suffered from undoubted ague, but in the remainder no evidence or suspicion of ague or malarial poison existed.

The course of the disorder is an interrupted one. The paroxysms may recur with more or less regularity for a period of a few days, or five or six weeks, and then cease altogether for a few days or weeks, or months, and recur again for a period as before. In this way it may continue an interrupted course for many months or years; in one case for so long a term as eleven years.

The prognosis is generally good. Of the twenty cases mentioned above none died, twelve were reported as having completely recovered, one was convalescent, and seven were still in progress when reported. By complete recovery is understood that there was no recurrence of the paroxysms for a period varying from six weeks to four years. It must, however, be borne in mind, that a relapse may take place after a pause of several months—in one case a recurrence took place after a pause of five months.

The following three examples will serve to illustrate the general course and symptoms of the disease. The first and second were observed by myself, the third by Dr. Ritchie; these cases have not been elsewhere published:

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[1] Berl. klinisch. Wochenschr., 1881, p 42.

CASE 1.—J. J., an iron moulder from Stockport, æt. 23, consulted me in March, 1868. He was thin and weak, with a conspicuously white, pallid countenance. He stated that for a period of twelve months he had been in the habit of passing dark bloody-looking urine from time to time at frequent intervals. His complaint began in the following manner: He was standing in the street one cold afternoon in March of the previous year, when he was seized with chilliness and shivering and pain in the loins. When he got home he voided urine, and was surprised to see it of a dark-red color. From this time up to the date of his visit to me he continued, with only short intervals, to pass every day urine of a similar character. He became so weak that he did not leave his house for ten months; but he never took to his bed, except for a day or two.

The discharge of the dark urine was essentially paroxysmal and intermittent. He would go on passing bloody urine daily for periods varying from three to six weeks—then the urine would be natural for an interval of three, four, or even ten days, and then become bloody again for a stretch of several weeks. But the urine was never constantly bloody during any whole period of twenty-four hours. During each diurnal circle, three paroxysms occurred with great regularity: one in the morning (after breakfast) between nine and ten, a second between two and three in the afternoon, and a third between six and nine in the evening. Each paroxysm presented the same succession of symptoms. It began with chilliness, which speedily went on to shivering so that his teeth chattered—at the same time there was severe pain in the loins and hips. At the end of twenty or thirty minutes these symptoms wore off, and then he felt a desire to void urine—and the urine then passed was always bloody; this completed the paroxysm. It was never followed by a hot or sweating stage. If he made water between the paroxysms, the urine was either perfectly clear and natural, or only slightly dark. The urine voided before breakfast was always quite natural.

The patient visited me several times, and brought me several specimens of the bloody urine. On one occasion he voided some in my presence, and a description of this will answer substantially for all the rest. It was of the color of the darkest port wine—so dark as to be almost opaque, except in thin layers; it corresponded pretty closely to No. 9 on Vogel's scale. A brownish deposit formed on standing; sp. gr. 1028; it was highly albuminous, and faintly alkaline. On boiling, the albumen coagulated into a chocolate-colored clot, but the supernatant fluid did not lose its black-red color. The deposit consisted of a granular matter. It contained numerous crystals of oxalate of lime; but no blood-disks could be recognized.

In April I admitted the patient into the Infirmary, with a view of studying his case more fully. But, with the exception of the first morning, the urine was perfectly normal throughout his stay, and he went home in a week. While in the Infirmary his blood was examined under the microscope, but nothing unusual was found. The liver and spleen were thought to be somewhat larger than usual—otherwise all the organs were healthy. After leaving the Infirmary, he continued to attend as an out-patient for some months—taking constantly, three times a day, a pill containing three grains of quinine and one grain of sulphate of iron. Under this treatment, the symptoms gradually subsided, the paroxysms became slighter and slighter, and at the end of three months ceased altogether. The general health also greatly improved. After the cessation of the paroxysms, he began to suffer from subacute rheumatism, with swelling and pain in the joints. He was troubled in this way for more than a twelvemonth, and went to Buxton, where he derived great benefit, and finally recovered. I saw this man a few days ago (March, 1870); there had been no return of the paroxysms, and he looked well and ruddy.

The patient stated positively that, except at the first onset of his complaint, the paroxysms came on independently of exposure to cold; he was just as bad in the summer as in the winter. Throughout his illness he ate and slept well. He had never lived in an aguish district, and he still occupies the same house as when his ailment commenced.

CASE 2.—The notes of this case are imperfect, and my own observations are confined to an examination of the urine. On November 28, 1871, my colleague, Dr. Renaud, sent me two samples of urine which had been voided by the same man at different periods of the same day. The contrast between the two samples was marvellous. One sample was of the usual yellowish-amber color, clear and free from a trace of albumen—in short, perfectly normal. The other was of the blackest red color, quite opaque, except in the thinnest layers. Its sp. gr. was 1032; it was so albuminous that it solidified into a chocolate-colored jelly when boiled in a water-bath. On standing, it deposited a copious dark-brown sediment. Under the microscope (see Fig. 33) this was found to consist of amorphous granular matter, amid which were seen numbers of tube-casts. The casts were mostly of a medium size, and dark granular character. Some were small, and a few almost transparent and approaching the hyaline character. Not a single recognizable red blood-disk could be seen, but a few corpuscles, resembling the white blood-globules, were scattered here and there. The field was full of minute bright specks of oxalate of lime octahedra. The proportion of urea, as ascertained by Liebig's method, was 5.2 per cent.

When the urine was boiled in a test-tube, the brown albuminous clots separated, and left a dark amber transparent supernatant liquor.

The history of the case, as far as it was gathered by Dr. Renaud, was as follows: The patient was a man of forty, of a sallow complexion, who felt, nine weeks before, as if he had taken cold. A fortnight since he lost his appetite, and shivered, and afterwards perspired. He kept his bed four days. On November 21st he passed, at noon, urine the color of blood; and he has done so nearly at the same time (*i. e.*, once in twenty-four hours) till yesterday. At other times of the day he passed urine of the natural color. I have not been able to obtain further particulars of this case.

CASE 3. (From the notes of Dr. Ritchie.)—T. M., a tailor, married, æt. 32, consulted me on December 18, 1869. He was above the medium height, dark-complexioned, and had a sallow appearance. He gave the following account of himself: He had formerly been a soldier, and had lived for nine years in the West Indies; during that time he had suffered from "black fever," and from repeated attacks of ague, the last of which seized him towards the end of 1865. These were the only illnesses he could remember till March, 1869, when he reminded me he had been under my care in the out-patients' room of the Manchester Royal Infirmary, suffering from pleurisy with limited effusion; and again, in September, 1869, from subacute rheumatism.

He now complained of passing bloody-looking urine once or twice a day—that passed in the intervals being apparently quite healthy. He first observed this symptom about two years before, under the following circumstances: He had been engaged in some gymnastic exercise one November evening, and feeling greatly overheated, had imprudently gone to the door of the gymnasium to cool himself, when he was suddenly seized with chilliness and violent shivering, followed by severe pain across the loins, and a feeling of nausea. About an hour afterwards he voided a small quantity of urine having the color of porter. The urine passed the next morning before breakfast was apparently quite natural, but at night the porter-like color was again present. For about two months the discharge of this dark urine took place once or twice every day, and then spontaneously ceased. He had had two similar attacks since—one in March, 1868, lasting about six weeks, and the other in September of the same year, lasting nearly four months.

His present attack, which was the most severe he had yet suffered from, began about a fortnight before he came to see me. It immediately followed exposure to cold and wet, and was ushered in by all the premonitory symptoms which characterized his previous seizures. The phenomena observe the following order: He feels perfectly well on getting out of bed in the morning, and usually passes about ten or twelve ounces of straw-colored urine; about ten o'clock he begins to yawn and shiver, and feels "as if he couldn't stretch himself enough;" he then suffers a dull, heavy pain across the loins, which sometimes extends round to the umbilicus, passing down the thighs; the testicles become retracted. There is no pain along the course of the ureters. The shivering and lumbar pain increase for about an hour, at the end of which he usually passes from eight to ten ounces of porter-like urine, upon which the symptoms gradually disappear. He usually voids urine again about one or two o'clock, which to all appearance is perfectly healthy. About four, the same succession of phenomena runs its course, to be followed by the discharge of dark urine about five, and again about ten o'clock in the evening. He has thus had three distinct paroxysms every day from the 2d to the 15th of December—remaining perfectly free from them between the night of the 15th and eleven o'clock in the forenoon of the 18th, when he began again to pass the porter-like urine. He stated that he usually went to bed when he felt the paroxysm coming on, and that he had the greatest difficulty in keeping himself warm; "so different," as he remarked, "from what it used to be, when he would have the ague."

It was about 2 P.M. on the 18th when he came to see me, and he was requested to send for me on the occurrence of the next paroxysm. A message was received from him about 9.15 the same night, and half an hour afterwards I was with him. He was in bed, shivering violently, and feeling very cold; his temperature in the axilla was 96.6° Fahr.; he suffered from the lumbar pain and feeling of sickness previously described; the testicles were closely retracted. He was ordered a warm drink, and to have an additional pair of blankets on his bed.

A few minutes after my arrival, he passed a quantity of urine, which was secured for the purpose of examination. Five minutes afterwards, he said that he was perfectly free from pain and discomfort, and felt quite well. His temperature was then 98.6°. There was slight tenderness over both kidneys on deep pressure, and also over the eleventh and twelfth dorsal vertebræ. Lungs apparently healthy, with the exception of slight comparative dulness over the right back—no doubt the remnant of the pleurisy from which he had suffered in the preceding March. Heart sounds and rhythm normal. Liver measured five inches vertically in the mammillary line. Spleen nearly three and a half inches; colorless blood-corpuscles slightly, but not markedly, increased. His general health was good; appetite fair; bowels had not been relieved for two days.

The urine which he was seen to pass measured eight ounces; it was not unlike porter in color, but had a redder tint; it was almost opaque—neutral or feebly alkaline in reaction; sp. gr. 1026. It contained a large amount of albumen which, on the application of heat, was thrown down in the form of a chocolate-colored clot, increased on the addition of nitric acid; the coagulum occupied about half the bulk of the urine examined, the supernatant fluid retaining its intensely dark reddish-brown color. On microscopic examination, it was found to contain abundant octahedral crystals of oxalate of lime, a few granular tube-casts, a large quantity of granular matter of a dark brownish-red color, and a quantity of amorphous urates. No blood-corpuscles could be detected.

The urine passed on the morning of the 19th December, before breakfast, possessed the following characters: it measured about 10½ ounces, was of a light amber color, acid, sp. gr. 1018; contained no albumen; on standing, the dense, snow-white, cloud-like deposit, characteristic of the presence of oxalates, was thrown down. Under the microscope, it was seen to contain octahedral crystals, pavement epithelium, and granular-looking cells.

The usual course of the paroxysms was observed, viz., that porter-like urine was voided three times a day, preceded by the same succession of symptoms. He was first ordered ten-grain doses of gallic acid three times a day, then tannic acid, and afterwards turpentine, without the slightest change in his symptoms. On the 28th December, he was ordered two pills three times a day, each containing sulphate of quinine 3 grains, sulphate of iron 1 grain, and strychnia $\frac{1}{60}$ grain. Under this treatment he gradually improved, the paroxysms coming on less frequently, and being milder in their character, till the 17th January, 1870. He remained perfectly free from that date till the 5th of April, when he had another similar attack after a sudden chill. The paroxysms on this occasion were slighter, and only came on twice daily, viz., in the forenoon and at night. The urine presented the essential characteristics described fully above. He was placed on the same treatment which had proved successful on the former occasion, and the paroxysms ceased entirely on the 14th of April.

I had lost sight of this patient till the 31st March, 1871, when a letter was received from him, stating that he had gone to reside in the north of Ireland, and that he had remained perfectly free from his former attacks from the time I had seen him last till the beginning of that month. He had then suffered for nearly a fortnight a recurrence of his former complaint, from which he had just recovered under the use of the same treatment that had been previously adopted.

ETIOLOGY.—The liability to paroxysmal hæmoglobinuria seems to be almost exclusively confined to males, one only of twenty collated cases occurring in a female. The *age* of the patients at the time of invasion ranged from 2 years of age to 48; two cases being under 20, seven between 20 and 30, six between 30 and 40, two between 40 and 50, and three cases in which the date of invasion is not specified. A hereditary tendency has been occasionally noticed.

Hæmoglobinuria has been known to occur occasionally in the course of chronic Bright's disease.

As to the *exciting cause* of the disease, in two cases out of the above twenty it was found distinctly connected with malarial poisoning, both patients actually suffering from ague at the time the hæmoglobinuria was first observed. In all the other cases (with one exception) the disease was clearly attributable to vicissitudes of temperature or exposure to wet. The effects of exposure to cold are well exemplified in a case of Dr. Johnson, cited by Dr. Dickinson (loc. cit.), in which the patient, so long as he remained in bed, continued free from the paroxysms; but if he sat up and got chilled, a paroxysm came on. In a case mentioned by Dr. Pavy ("Path. Soc. Trans.," vol. xviii. p. 157), the patient had sometimes averted an attack by going indoors directly he felt it coming on, and sitting before the fire and drinking something warm. Sir W. Gull believes there is reason for thinking that a blow or injury to the loins may be the cause of this affection; and cites the case of a young lady who in getting into a railway carriage fell and hurt her back, shortly after which she passed dark bloody-looking urine, in which he found, on careful examination, no blood-corpuscles, but only the granular pigment matter of disintegrated blood-corpuscles. Whilst admitting the possibility of such injuries causing hæmaturia, or even as in this case hæmoglobinuria, we should, however, in the absence of further evidence on the point, hesitate to accept them as a cause of *paroxysmal* hæmoglobinuria.

Fleischer[1] has recorded a case in which heat and cold produced no effect, but the paroxysms were always brought on by exercise. In the rare cases which occur in women, Wolf[1] saw the attack brought on under the influence of menstruation alone. Hæmoglobinuria has been noticed in cases of Raynaud's symmetrical gangrene. (*See* Southey, "Clin. Trans.," vol. xvi. p. 167.)

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[1] Berlin. klin. Wochenschr., 1881, No. 47.

Murri asserts that a causal relation exists between syphilis and paroxysmal hæmoglobinuria. He found a history of syphilis in such cases, and obtained a cure by the use of antisyphilitic remedies.

THE PATHOLOGY of the disorder is very obscure. It very rarely ends fatally, and the few post-mortem examinations which have been obtained have given no clue to the origin of the disease. More important evidence has resulted from a consideration of hæmoglobinuria produced experimentally in animals, and from a close examination of the paroxysm as it occurs in man.

Ponfick found that the hæmoglobinuria caused by transfusion (p. 157) was accompanied by symptoms closely resembling those of the paroxysmal affection in man. In the artificial affection, however, he found that there was a solution of hæmoglobin in the blood-serum, and that the red blood-corpuscles showed peculiar changes, which he believed were produced by their partial destruction. Very similar changes have been noticed in the paroxysmal affection of man. If during the paroxysm the serum obtained from a blister or a cupping-glass be examined, in many cases, at least, it will be found to contain hæmoglobin in solution (Küssner,[2] Hayem, and others), and presumably the whole of the serum of the body is in the same condition. The microscopic examination of the blood (p. 160) during the paroxysm has also revealed changes similar to those described by Ponfick in the artificial affection. It therefore seems probable that in paroxysmal hæmoglobinuria we have to deal with a condition in which the blood-corpuscles give up their hæmoglobin to the surrounding serum.

We know, however, from the researches of Bernard and Stokvis, that if certain forms of albumen other than serum albumen circulate in the blood, they are usually filtered off by the kidneys without change, and appear in their natural condition in the urine. So then, if a solution of hæmoglobin circulate in the blood, it also will be excreted in the urine.[3] Most observers are now agreed that the solution of the hæmoglobin in the serum precedes its appearance in the urine, and that the symptoms of kidney affection, which are sometimes present, are due to the irritation produced by the passage of the hæmoglobin through those organs. Adams[1] found that the glomeruli of the kidney alone were concerned in the excretion of the hæmoglobin. In Dr. Dreschfeld's case of poisoning by chlorate of potash (p. 157), hæmoglobin could not be seen in the glomeruli, but only in the convoluted tubes, while Ponfick[2] has lately shown that all the secreting parts of the kidney may be concerned in the process.

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[1] Centralbl. f. Med. Wissensch., 1883, p. 820.

[2] Deutsch. Med. Wochenschr., No. 37, 1879.

[3] If only a small amount of hæmoglobin be dissolved in the serum, it may appear in the urine as bile-pigment (Cohnheim).

A few observers, and amongst them Rosenbach and Lépine, still hold that the kidneys are primarily affected, and that in them the blood-corpuscles are destroyed, and their hæmoglobin reabsorbed before it can circulate in the blood-serum. Rosenbach mainly based his opinion on the fact (*see* p. 160) that he observed albumen to appear in the urine before hæmoglobin. There are, however, only few observations in which such disorder of the kidney function was found; while Roux,[3] working in Cohnheim's laboratory, has shown that the amount of albumen found in the urine of an ordinary case is just sufficient to combine with the amount of iron present, in the proportions necessary to produce hæmoglobin, and that hence the albumen present is probably derived entirely from the hæmoglobin.

The behavior of the blood-corpuscles has been investigated by Ehrlich and Boas, by ligaturing a finger and then placing it in ice-cold water. In a healthy person this produced no change on the blood of the finger, but in a person who was subject to hæmoglobinuria, solution of hæmoglobin in the serum was observed, and also the changes in the blood-corpuscles described by Ponfick were seen. The red blood-corpuscles were, therefore, less resistant to cold than in the normal state. Boas also showed that they were more easily destroyed by the electric current than were healthy corpuscles.[4] It seems probable that a similar destruction of the blood-corpuscles may take place in the exposed and chilled parts in the paroxysmal affection, and such a view is supported by Dr. Southey's cases of symmetrical gangrene already mentioned.

The cause of this condition of the blood-corpuscles is, as yet, a matter of pure speculation. According to Murri the cause is to be sought in a diseased condition of the blood-forming organs, which rendered the corpuscles less resistant to cold and to carbonic acid than normal corpuscles are. Whether primarily or secondarily, however, the nervous system must be an active agent in producing the phenomena of the paroxysm.

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[1] Dissertat., Leipzig, 1880.

[2] "Verhandl. des Congresses f. inn. Medic.," Wiesbaden, 1883. This paper may also be consulted for information concerning the so-called "Hæmoglobinæmia," which precedes the change in the urine. The author shows that not only the kidneys, but also the spleen and the liver take part in removing the hæmoglobin from the circulation.

[3] Cohnheim's Allgemeine Pathologie, ii. p. 295.

[4] Only one experiment, however, was successful, and further evidence is desirable.

There seems to be some connection between this affection and ague, but its precise nature is as yet unknown. Though related, they are not identical, as in by far the greater number of cases there has been neither an aguish tendency nor any evidence of exposure to malarial influences.

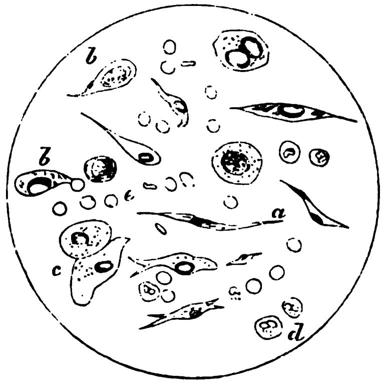
TREATMENT.—The remedies appropriate to the ordinary forms of hæmaturia have been found wholly inefficacious in this disorder. In two of the reported cases the attacks seem to have passed off without any medicinal treatment, simply by avoiding exposure to cold. In one case, recorded by Dr. Dickinson, cupping over the loins, vapor baths, gallic acid, quinine, iron in various forms, were tried in succession, but nothing seemed to affect the disorder; "the hemorrhage always ceased on the removal of the cold which caused it." This patient had an intercurrent attack of pneumonia, after which he passed into a typhoid condition, from which he slowly recovered under the use of stimulants, and afterwards of quinine and iron, and was discharged well. Dr. Harley gave mercurials, and afterwards quinine in his cases, with marked benefit, one patient having remained free from the paroxysms for four years, during which he was under observation. Sir W. Gull gave two drachm doses of compound tincture of cinchona three times a day, with benefit; the patient went out of hospital "convalescent." Dr. Hassall found that the hemorrhage was considerably restrained, by giving, night and morning, a powder containing tannic and gallic acids, and burnt alum, with a mixture containing quinine, sulphate of iron, and excess of sulphuric acid during the day. Dr. Habershon found that quinine and arsenic, and Dr. Greenhow that quinine and perchloride of iron, and afterwards quinine and syrup of the iodide of iron, with iodide of potassium, had the effect of causing the urinary symptoms to subside, and the patients remained free from attacks for several months afterwards, during the time they were under observation. Dr. Beale calls attention to the importance of giving quinine in full doses, not less than six grains, in order to combat the disorder successfully. Dr. Begbie, in one of his cases, found that the paroxysm did not recur when the patient took twenty grains of sal ammoniac three times daily. During the paroxysm, Dr. Ritchie found the best treatment was to send the patient to bed, apply artificial heat, and administer warm stimulating drinks, such as hot brandy and water. The evidence generally is strongly in favor of quinine and iron as the most effective medicinal agents. In cases where a syphilitic taint is suspected, it would be well, in view of Murri's results, to prescribe an anti-syphilitic treatment.

VII.—CANCEROUS AND TUBERCULOUS MATTER IN URINE.

When cancer or tubercle of any part of the urinary tract has gone on to ulceration, the urine carries away with it some of the disintegrated elements, giving rise to an amorphous-looking grumous deposit. Sometimes masses of the morbid tissue as large as a horse-bean are discharged with the urine, and more or less blood is always mixed with such deposits.

Very great caution is requisite in coming to a conclusion as to the *cancerous* nature of cells found in urine, on account of the great similarity between the irregular transitional forms of the epithelial cells lining the urinary passages, and the cells of cancerous growths. Indeed it would be quite unsafe, in such a case, to rely on the mere form and size of individual cells. In the annexed drawing (Fig. 34) may be seen the diverse shapes discharged with the urine in a case of malignant fungus of the bladder. If the forms be compared with those in Figs. 23 and 31 (*g*), the similarity of the cells will appear very striking. It is more safe to take the entire character of the deposit into consideration. It may be described as a thick, dirty, blood-stained sediment, containing abundance of blood-corpuscles, mixed with spindle-shaped, oval, and irregular cells. Pus-corpuscles may be either wholly or nearly absent. The presence of shreds or pieces of solid tissue appreciable to the naked eye, should be carefully looked for: their occurrence is almost a certain proof of the existence of some morbid growth. The character of the deposit generally, and especially the presence of numerous

FIG. 34.



Cell from the urine of a woman with fungus of the bladder. *a*, Fibro-plastic cells; *b*, *b*. Cancer cells; *c*. Epithelial cells; *d*. Pus; *e*. Blood.

spindle-shaped (fibro-plastic) cells, which cannot be mistaken for epithelial elements, indicate clearly that some morbid growth or natural tissue is being broken up. The collateral symptoms are then generally sufficient to decide whether the broken-up tissue is a portion of the natural membrane or an adventitious growth. In cancer of the kidney no help to the diagnosis must be expected from the character of the urinary deposit (*see* Cancer of Kidney).

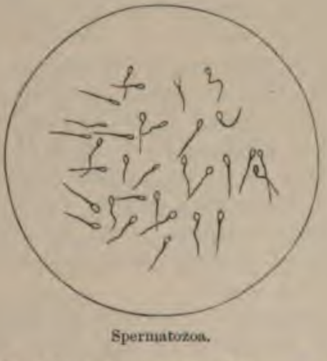
The discharge associated with *tuberculous* ulceration differs from that of a cancerous fungus in being largely purulent; indeed, pus-corpuscles are usually the chief appreciable formed elements in the urine in cases of tubercle of the kidney and bladder. But in other cases, broken-down cheesy masses may be seen, together with a large quantity of amorphous, or barely morphous granular débris (for the discovery of tubercle bacilli in the urine, *see* Tubercle of the Kidney).

It follows, of course, that cancerous and tuberculous masses may exist in the kidney, or beneath the mucous membrane of the urinary passages, without contributing anything to the stream of urine. It is only when ulcerated that their elements escape with the urine; before this takes place they may, however, give rise to copius and oft-repeated hemorrhage.

VIII.—SPERMATOZOA IN URINE—SPERMATORRHŒA.

The admixture of semen with the urine gives rise to a mucous-looking deposit. When in large quantity, white albuminous flakes and masses are seen; these exhibit a viscid consistence when taken up with the pipette. The microscope reveals the existence of spermatic filaments, consisting (Fig. 35) of a minute oval head, not more than $\frac{1}{10000}$ of an inch in breadth, and a long whip-like tail of extreme delicacy. The length of the entire filament is $\frac{1}{600}$ of an inch.

FIG. 35.



Spermatozoa.

When freshly shed, and still living, they exhibit active eel-like movements, strongly suggestive of volition;[1] but as seen in urine they are always motionless. They offer considerable resistance to disintegration, and may sometimes be recognized in decomposed urine which has been kept for weeks.

A certain quantity of seminal fluid necessarily finds its way into the urine of both sexes after coitus; also into the urine of men after involuntary nocturnal emissions.

Involuntary nocturnal emissions occurring occasionally in the young and continent, are not to be regarded as within the limits of disease; but when they take place two or three times weekly or oftener, or when the acts of defecation and micturition are frequently followed by a glairy discharge, a diseased state must be acknowledged to exist; and one also, as experience proves, exceedingly difficult to deal with. Whether it be that the mental phenomena observed in these cases are altogether secondary to the genital defect may well be questioned; but it is an important—indeed *the* important—fact in relation to involuntary seminal discharges, that they are associated with a deplorable state of mind. Much of this is, no doubt, owing to the prurient eagerness with which persons so afflicted seek satisfaction to a fatal curiosity, in the publications of unprincipled quacks, who lure their victims with libidinous descriptions, and afterwards terrify them with exaggerated and lying pictures of the fate which awaits them.

But there is a danger that the legitimate practitioner may come to look upon cases of this class too lightly, and thus be the indirect occasion of their seeking the help which is their injury.

The least serious cases are those in which the emissions are solely nocturnal. As long as the complaint is confined within these limits the general health does not suffer, and the mental state is seldom gravely disturbed. Sometimes, however, individuals of fervid imagination, whose health is from any cause below par, fix upon this incident (nocturnal emissions) with obstinate tenacity, and hinge their ill-health entirely upon it, when in reality it has nothing to do with the matter. Persons go on for years subject to nocturnal pollutions without any harm resulting, but when they chance to become dyspeptic, or their nervous system becomes upset by overwork, *then* these emissions loom largely to their imaginations, and they connect them with their failing health.

When seminal discharges occur daily, and accompany or follow defecation and micturition, a greater departure from the natural state is betrayed; and it is seldom that such a state of things continues for any length of time without inducing pallor, weakness, want of zest and energy for work, as well as a fidgety, vacillating, and sometimes very depressed state of mind. Nevertheless, these consequences frequently altogether fail. There was recently a patient under my care at the Royal Infirmary—a ruddy, strong-looking young man of six-and-twenty—who had been in the habit, according to his own account, for the last seven years, of discharging large quantities of seminal fluid almost daily, more especially with micturition. In a specimen of his urine brought to me, there was at least a tablespoonful of glairy matter having the microscopic and other characters of semen. The mental state was certainly shaken, but solely, as it appeared to me, from the diligent study of Mr. Dawson's book on spermatorrhœa. He talked with a sort of gloomy satisfaction of being tired of life, but it was with an air as if he were repeating a lesson, and not as one revealing a terrible conviction.

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[1] Students may be reminded that spermatozoa are not really independent animals, but simply the escaped contents of a cell. They are floating cilia, and resemble the oscillating sperm-cells of the antheridæ of mosses.

The type of mental disturbance usually associated with spermatorrhœa, is common in this as in other large towns, independently of seminal losses, among persons—chiefly men of business—whose health has given way from too engrossing application to exciting pursuits. Such persons become nervous, apprehensive about themselves to a distressing degree, pusillanimous, subject to attacks of incomplete syncope; they lose their sleep and sometimes their appetite; there is some real emaciation and a great deal of fancied wasting. They pour into the ears of their medical attendants an endless variety of symptoms, and worry them beyond the most tedious hysterical women. Such patients, although often men of middle age, or at least beyond their first youth, and fathers of families, rarely fail to complete the catalogue of their ailments with a reference to what they conceive to be some anomaly of their sexual functions.

Involuntary discharges are not confined to youth or middle age. Men advanced in years are sometimes tormented in the same way, and exactly the same state of mind is observed in them. They imagine their "substance" to be ebbing from them, and their virility departing. A gentleman over sixty years of age, the father of a family of married daughters, was so concerned about a slight seminal discharge which in no way affected his health, that he forwarded to me for examination over a hundred specimens of his urine.

In the TREATMENT of this class of cases, the first point to establish is whether the trouble of the nervous system is the primary phenomenon, and the disturbance of the sexual functions only an insignificant incident, or whether the seminal losses are in such frequency and quantity that they may be regarded as having a hand in evolving the symptoms complained of. The greater majority of cases belong to the former category; and indications for treatment are to be looked for in the general state of the patient and the circumstances surrounding him, rather than in the condition of the sexual functions. If it appear, after a patient sifting of the actual phenomena and the past history of the case, that the seminal emissions must be regarded as the fundamental ailment, the next point is to inquire into the existence of any local cause for the emissions. The irritation of ascarides or hemorrhoids sometimes occasions involuntary discharges: also herpetic eruptions about the prepuce. Lallemand enumerates a long prepuce as contributing to the same, by the lodgement which it affords, in uncleanly persons, to offensive secretions. Whatever be the local cause discovered, its immediate removal is of course the first step in the treatment.

In the absence of a local cause, the evil can usually be traced to venereal excesses, masturbation, and the reading of salacious literature. Some of these cases are very difficult to deal with. An attempt must first be made to put a stop to the practice which is the cause of the complaint. The further treatment should be directed to improving the tone of the muscular system by daily ablutions with cold water or brine, by sea bathing, regulated exercise, change of air, etc. The state of the patient's mind often requires that the time, quantity, and material of the meals shall be minutely regulated. The diet should be nourishing and bland; spices and condiments should be avoided. Malt liquors and the lighter wines are to be cautiously employed; the quantity must be judged by their effects. Any quantity which produces flushing of the face is too much. An opiate sometimes renders good service by securing a good night's rest. Astringent and ferruginous tonics offer valuable aid to the hygienic treatment. Tincture of the muriate of iron has appeared to me to produce a better effect than any other preparation. A blister to the perineum has sometimes seemed to diminish the emissions. In cases of inveterate masturbation, Mr. Hilton found that he could invariably put a stop to the practice by applying a strong solution of iodine or blistering fluid to the penis so as to render the organ too sore for manipulation.[1]

Lallemand recommends the local application of nitrate of silver to the orifices of the ducts of the vesiculæ seminales by means of his porte-caustique. I cannot say that I have ever seen cases in which this severe proceeding seemed justifiable. It must be remembered that it is not without danger. Dr. Bird relates an instance in which a dangerous cystitis was produced in a healthy person by the local application of the solid nitrate of silver in this manner. Dr. Chambers has communicated another and more untoward example, in which death followed the application of an irritant ointment by means of a catheter in a case of imaginary spermatorrhœa.[1]

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[1] Lancet, 1863, II. 123.

Dicenta, B. Schulz,[2] and Benedikt,[3] speak in high terms of the constant galvanic current. Schulz directs the current to be transmitted along the vertebral column for one or two minutes, and repeated three or four times a week. Twenty or thirty Daniel's elements, of medium size, should be used; the positive pole should be applied to about the fifth dorsal vertebra, and the negative to the sacrum or perineum. My colleague, Dr. Dreschfeld, has published an account of three cases successfully treated by the application of the constant current twice a week to the lumbar region. ("Practitioner," 1874, p. 360.)

IX.—MICROÖRGANISMS IN THE URINE.

The microörganisms met with in the examination of the urine may be classed into three categories—namely: *Torulaceous Vegetations*, *Sarcina*, and the various forms of *Bacteria*.

1.—Torulaceous Vegetations (Saccharomyces).

Torulæ appear in the urine after emission only. They have not been detected in the perfectly fresh secretion—but are exclusively derived from germs which gain access to the urine and grow in it after it has left the urinary passages. They appear at first as minute oblong cells (sporules), either lying separate, or strung together into short chains. Presently they elongate into transparent hollow threads which divide and interlace into a fleecy cloud (thallus). The most common are the sporules of the blue and brown moulds (*Penicilium glaucum* and *Aspergillus niger*) and of the yeast plant (*Saccharomyces Cerevisiæ*). After a few days' growth the two former ascend to the surface and form patches of mould, constituting the aerial fructification of these vegetations. Torulæ are, strictly speaking, extraneous impurities in the urine; and they are only of importance from their liability to be confounded with blood-corpuscles or other objects derived from the urinary passages. Torulæ are distinguished from blood-disks by the great difference of size among the individual cells (Fig. 36); the presence of a nucleus in the larger sporules, their tendency to assume an elongated or oval form; and the indications of budding and commencing formation of a thallus. Torulæ appear in the urine sometimes in a few hours after emission—more commonly after the lapse of a day or two.

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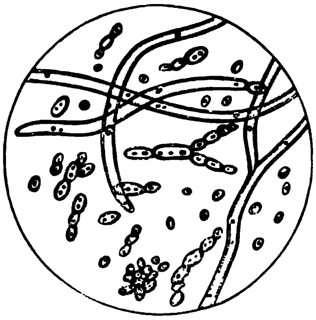
[1] Lancet, 1861, p. 582.

[2] Year Book, 1863, p. 300. Reports on Surgery also.

[3] Elektrotherapie, Vienna, 1868, p. 447.

Fig. 36.

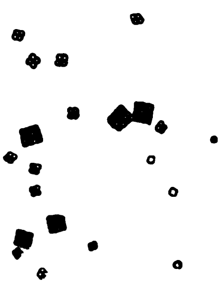


Torulæ in urine.

They require an acid reaction for their free growth; and they cease to multiply, and finally perish, when the urine becomes ammoniacal.

2.—Sarcina.

Fig. 37.[2]



Sarcinæ in urine.

Since Heller and Mackay, in 1848, first discovered sarcinæ in urine, they have been observed by Johnson, Beale, Welcker, Munk, Begbie, and myself. The seat of production of this vegetation is probably the bladder; and it is discharged with the urine, sometimes in great quantities, and forms a grayish-white amorphous-looking deposit. It consists of the same elements as the sarcina ventriculi (of Goodsir), and is usually regarded as the same species. Both the cubical masses and their component particles are, however, smaller than those of the gastric sarcina, and Rossmann and Welcker[1] consider these differences sufficient to establish a specific distinction. It seems more probable, however, that the differences in the habitat and conditions of growth are sufficient to account for the diversity of size. Dr. P. Munk[3] has shown that one of the points relied on by Welcker, namely, the absence (in urinary sarcina) of cubes containing more than 64 particles, is not constant. Munk found cubes of 512 particles. In some vomited matter sent to me for examination by Dr. Scowcroft, of Southport, I detected small-sized sarcinæ mixed with those of ordinary dimensions.

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[1] Ueber Sarcina im Urine des Menschen. Henle and Pfeuf. Zeitsch. 3tte R. Bd. V. 199.

[2] After Welcker, Henle and Pfeufer's Zeitsch., Bd. V. Taf. x.

[3] Ueber Harnsarcine—Archiv f. Path. Anat. 1861, p. 570.

This curious vegetation is generally associated with some disorder of the urinary organs (renal pains, painful micturition, vesical catarrh, etc.). It grows, or at least exists, both in acid and ammoniacal urine. In Munk's case the fungus grew in great quantities during the summer months, and disappeared almost wholly in the winter months; and this was the more remarkable as the patient (who was paraplegic) kept his bed continuously from year to year. Dr. Begbie's patient[1] suffered from lumbar pains and frequent micturition, together with hypochondriacal and dyspeptic symptoms.[2] In the case seen by me, the patient—a merchant about sixty years of age—was suffering from long-standing chronic cystitis, due to enlarged prostate.

No treatment yet tried has had any appreciable effect in checking the growth of sarcinæ in urine.

3.—Bacteria (Bacteruria).

Urine, like other organic fluids, when exposed to the contact of air-dust or of ordinary water, passes sooner or later into a state of decomposition. It is then found to swarm with bacteria. The organisms under these circumstances gain access to the urine, and grow in it after it has left the body. But there are also conditions in which the urine contains bacteria at the moment of emission. In these cases the organisms must have grown and multiplied in the urine during its sojourn in the urinary passages. These cases may be conveniently embraced under the general heading of *Bacteruria.* But inasmuch as the bacteria discharged with the urine are of various kinds or species, and as the different species affect the urine in quite different ways, the resulting symptoms also differ greatly both in degree and in kind. Certain kinds of bacteria affect the composition of the urine very slightly, or not at all; others again rapidly break up its chief constituent, urea, into carbonate of ammonia, and thereby introduce into the previously bland secretion a fiery irritant, which is apt to light up a dangerous inflammation of the urinary mucous membrane. For these reasons it is necessary to divide cases of bacteruria into a certain number of groups or categories. How many such groups it may be eventually necessary to establish I cannot say—the subject is as yet new, and the field of inquiry only partially explored. The cases which have hitherto fallen under my observation appear to warrant a division into four groups; namely, (1) cases in which the presence of bacteroid organisms is associated with incipient putrefactive changes in the urine; (2) cases associated with ammoniacal fermentation of the urine; (3) cases in which some of the common forms of bacteria are present without decomposition of the urine; (4) cases in which micrococcus chains are voided with the urine. A good many examples of bacteruria are mixed cases—cases in which more than one form of bacteria coexist in the urine—but the above scheme of classification may be provisionally adopted; and I propose to devote a separate notice to each group.

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[1] Edin. Med. Journ. 1856-7.

[2] See also a paper by Heller—Wien. Med. Presse, xi. p. 13.

GROUP I. BACTERURIA ASSOCIATED WITH INCIPIENT PUTREFACTIVE CHANGES IN THE URINE.—This form of bacteruria is very common. The urine is more or less opalescent when voided; it is feebly acid, neutral or feebly alkaline. When examined under the microscope it is found to contain bacteria in active motion. The kinds of bacteria present in these cases are the common forms found in decomposing organic fluids, of which the least known is the Bacterium termo (Fig. 38). The urine on standing does not recover its transparency; on the contrary, the turbidity tends to increase, and the urine passes on pretty quickly to decomposition. This condition is accompanied by few or no symptoms; there may be a little heat about the genitals, or a slight undue frequency of micturition—but for the most part no complaint is made. This condition is not unfrequent in women of weak health suffering from leucorrhœa, and is common among men who have suffered from stricture, and who have frequently used catheters or bougies. The affection is in itself of no importance, and may persist for years without requiring attention; but it assumes a graver significance if, as I have reason to believe, it renders the subjects of it liable to the next form of bacteruria which is associated with ammoniacal urine.

GROUP II. BACTERURIA WITH AMMONIACAL FERMENTATION OF THE URINE.—This condition always involves the patient in serious suffering and danger. The change which occurs in the urine in these cases is the transformation of urea into carbonate of ammonia. The chemical nature of this change has been already explained (*see* p. 83). One molecule of urea with two molecules of water become two molecules of carbonate of ammonia, $CO(NH_2)_2 + 2H_2O = (NH_4)_2CO_3$. The transformation is an example of bacterial fermentation. Pasteur believes that the change is due to the action of a minute spherical bacterium to which Cohn has given the name of *micrococcus ureæ*. This organism consists of excessively minute round particles, lying free and in active movement, or strung together into short chains of two, three, or four elements each (*see* Fig. 38, *b*).

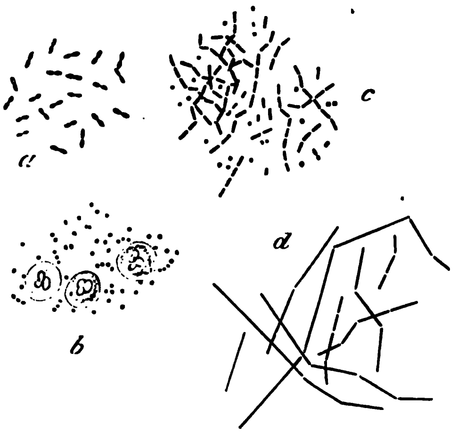
Ammoniacal bacteruria is apt to arise in old stricture cases, in cases of stone in the bladder, after operative procedures, whether lithotomy or lithotrity, in cases of enlarged prostate, paraplegia, morbid growths in the bladder, and in all conditions in which the organ is unable to empty itself completely, or which require the frequent use of instruments.

In these two groups of cases the offending organism gains access to the bladder by the urethra—at least in the overwhelming majority of cases. In the female the short and comparatively wide urethra offers obvious facilities to wandering bacteria to penetrate into the viscus from the external genitals. In the male the long and narrow urethra forbids this mode of entrance in the normal state. But in cases of gonorrhœa, or other type of urethritis, in which the passage is lined with a continuous layer of purulent discharge, it is quite easy to understand that along this purulent tract bacteria may breed their way up into the bladder. In a good many cases the infective organisms steal in with the instruments, which always come into use, sooner or later, in all kinds of vesical trouble. A dirty catheter is a most efficient infective agent. It must not, however, be overlooked that, in states of depressed vitality, septic germs may, occasionally at least, find their way into an ailing organ or tissue by the channels of the circulation. Such a mode of intrusion of bacteria-germs into the bladder in cases of paraplegia seems highly probable.

GROUP III. BACTERURIA WITHOUT DECOMPOSITION OF THE URINE.—Judging by my own experience, this is a condition far from infrequent. The organisms which are present in the urine in these cases are short moving rods and micrococci. I am not sure that the organisms are always of the same species, but they are evidently neither the bacterium termo nor the micrococcus ureæ, inasmuch as they produce no change in the chemical constitution of the urine. In some examples the organisms resemble the *Bacillus subtilis* (of Cohn), and the short rods are found accompanied with long slender threads (*see* Fig. 38, *c* and *d*).

The character of the urine in this group differs widely from that in the two previous groups. In decomposing urine (Groups I. and II.) the turbidity is persistent, and the organisms go on multiplying in it after it has left the body. But in the group now under consideration, the urine, although opalescent when voided, becomes clear on standing, and the organisms, together with the other formed elements (pus, etc.), subside to the bottom of the vessel. The supernatant urine continues transparent and acid for many days, and the organisms show no signs of multiplying. Indeed, the urine exhibits less tendency to decomposition than ordinarily healthy urine, and remains clear and acid for seven or ten days. Even when the urine is kept in the warm chamber at blood-heat the organisms do not multiply. All this leads to the inference that in this kind of bacteruria, the seat of growth of the organisms is not the urine itself, but some portion of the surface of the urinary mucous membrane.

FIG. 38.



Various kinds of bacteroid organisms found in the freshly voided urine. *a*. Bacterium termo; *b*. Micrococcus ureæ; *c* and *d*, other bacterial forms—not identified with certainty as belonging to any of the known species of bacteria.

The symptoms which are associated with this form of bacteruria are frequent and painful micturition, and pains about the neck of the bladder. They vary greatly in intensity—rising and falling apparently in unison with the increasing or lessening swarms of bacteria discharged in the urine.

This form of bacteruria seems to be controlled in an important degree by the internal administration of full doses (30 grains twice a day) of salicylate of soda.

In the last four years I have met with a considerable number of cases belonging to this group. The two following may serve as typical examples:

CASE 1.—A retired professional man about fifty years of age fell on his hip in February, 1881. As the hip continued painful, a strong solution of iodine was applied to it, which produced vesication. Four or five days afterwards there arose a violent irritation of the bladder. It was conjectured that this might be due to a congested state of the prostate, and on that view blistering fluid was applied freely to the perineum. This was immediately followed by an aggravation of the bladder symptoms. Micturition became excessively frequent and painful. These symptoms persisted with severity for a period of two months, and then began to abate under the use of warm baths. Neither blood nor albumen appeared in the urine during all this time, and the reaction of the secretion was always acid.

When he consulted me, three months after the accident, the same symptoms continued in a mitigated degree. He voided urine in my presence. It was opalescent, and swarmed with bacteria, but it was sharply acid. A portion set aside in a urine-glass became quite transparent in twenty-four hours and let fall a deposit consisting of bacteria rods mixed with pus-corpuscles. This urine remained transparent and acid for seven days in a warm room. Three other specimens subsequently examined behaved exactly in the same way. Thirty grains of salicylate of soda were prescribed to be taken twice a day. In less than a week the symptoms subsided, and the bacteria disappeared from the urine. In the subsequent year the symptoms returned, and continued with considerable violence for some weeks. The urine was again found to swarm with bacteria and to present the same characters as before. This second attack was also cut short in a few days by the salicylate of soda.

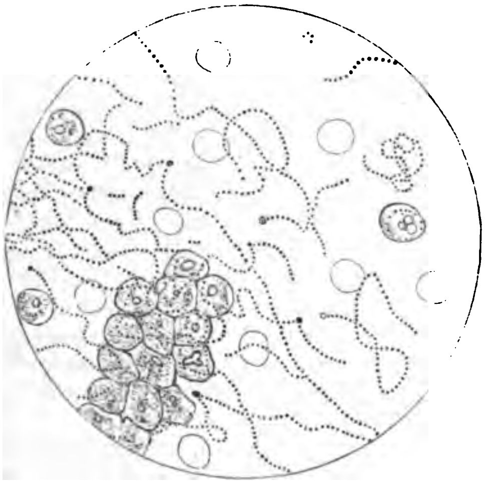
CASE 2.—A merchant, then aged 40, consulted me in 1881, suffering from severe vesical catarrh. The urine passed in my presence was found to be laden with actively moving bacteria, together with pus-corpuscles and a few blood-disks. (This man had suffered from cystitis ten years before, but in the interval had maintained fair health, although he had never been quite free from urinary trouble.) The reaction of the urine was acid; and it showed the same remarkable indisposition to pass into decomposition as in the preceding case. This man has visited me from time to time until the present year (1884). He has suffered from several recurrences of the bladder trouble — the urine on these occasions is always of the same character and the attacks are always relieved by the use of thirty-grain doses of the salicylate of soda. But the urine never becomes absolutely free from bacteria—and exposure to cold, worry, or excessive fatigue invariably brings on a recrudescence of the symptoms.

In neither of these cases had an instrument ever been passed into the bladder, and it must be regarded as probable that the organisms had originally obtained access into the bladder by the circulation. In the majority of cases of this group which I have encountered, instruments had been used at some time or other, and it was therefore impossible to be sure whether the organisms had not been introduced by their means.

A longer acquaintance with cases belonging to this group—that is, cases where bacteria-forms are discharged with the urine without there being any decomposition of the secretion—has convinced me that the organisms present in different cases are not of one uniform type, and that in some examples two or more distinct species are growing side by side in the bladder. The different microscopic character of the organisms is sufficient to substantiate this in some instances; but the microscope is a very imperfect guide in the study of specific differences among bacteria. More light will be thrown on the subject when the several kinds encountered in urine have been sifted by the method of artificial cultivation. In this branch of the inquiry I have made but little progress, and I should like to invite observers with more leisure than myself to enter the field.

GROUP IV. MICROCOCCUS CHAINS IN THE URINE WITHOUT DECOMPOSITION.—Although I have met with but one example of this group, the organism found in the urine was so distinctive that it warrants me in separating this form of bacteruria from the remainder. I have also succeeded in cultivating the organism artificially in a state of absolute isolation from all other organisms. The patient was a retired merchant, 68 years old. At the age of sixteen he went to Rio Janeiro and stayed there fifteen years. He then went to India for one year. For the last twenty-eight years of his life he resided in England. With the exception of a few slight attacks of gout he enjoyed good health until within three years of his death. At this period he began to suffer from recurrent attacks of hæmaturia. These attacks, at first slight and occurring at long intervals, became gradually more severe and more frequent. At length the bleeding became continuous; violent cystitis intervened and finally the patient died exhausted. After death three soft bleeding polypoid growths were found in the bladder. It is not necessary for the present purpose to enter more fully on the clinical history of the case nor to recount the various plans of treatment adopted. I first saw the case with my friend Dr. Ransome, of Bowden, in the spring of 1881, and we watched it closely until its termination in April, 1882. During the progress of the illness the urine had been repeatedly examined, with no other result than the finding of blood-corpuscles and leucocytes; but in the middle of July I detected something I had not observed before. I saw in the deposit a number of long delicate beaded threads. In all my experience of urinary examinations I had not seen anything like them in the fresh urine. After this date the urine was examined many scores of times, and the same beaded threads were invariably found in large numbers in every specimen. The urine was generally acid; and it showed no unusual tendency to decomposition. It contained no other organisms except the beaded threads until a late period of the case, when, as a sequence to the use of injections into the bladder, bacterium termo and the micrococcus ureæ made their appearance. This conjunction evidently hampered the growth of the beaded threads, and they nearly vanished from the urine before the termination of the case. The following account of these filaments was gathered from repeated examinations of different samples of the urine, and of the cultivated organism. To the naked eye the fresh urine looked very much like that from an ordinary case of acute Bright's disease. It had a smoky appearance, and deposited on standing a loose reddish-brown sediment. The sediment consisted of little soft brownish masses or flakes mixed with blood. Under the microscope these flakes were found to be composed of leucocytes or pus-like corpuscles intermixed with blood-disks. In these flakes the beaded threads were seen, twisted and turned in every direction, and forming an inextricable tangle of threads running in and out among the corpuscles. In perfectly fresh samples the filaments were found exclusively in these leucocyte-flakes; but after the urine had been kept awhile the flakes broke up more or less, and then detached fragments of the threads were seen scattered about in the field of the microscope. With a magnifying power of 500 diameters the threads were seen to consist of moniliform filaments of extreme delicacy and regularity of structure (see Fig. 39). Their width measured from a tenth to a fifteenth part of the diameter of a blood-disk. Their length varied greatly. Some were so long that they stretched right across and far beyond the field of microscopic vision; but they were for the most part so twisted and turned on themselves that it was impossible to gain a precise idea of their length. Under an immersion lens and with good illumination the filaments were resolved into a row of minute spheres apposed end to end like a string of beads.

FIG. 39.



Beaded threads—or micrococcus chains—from the freshly-voided urine of the case described in the text—mixed with leucocytes and blood-disks.

The organism was found to be easily susceptible of artificial cultivation, in either highly diluted albuminous urine or in filtered beef-tea. When a drop of the fresh urine was introduced into a sterilized flask containing beef-tea—and the flask was placed in the warm chamber at blood-heat—the new growth soon made its appearance. In from three to six hours light fleecy specks were seen scattered through the transparent medium. In the course of another hour or two these gathered themselves together into a voluminous soft cloud. After this no further change occurred—the process of growth seemed ended. From the first cultivation a second, a third, and so forth, up to a sixth cultivation were easily obtained in a state of perfect purity, and without exhibiting the slightest modification of form.

When a portion of the cloudy mass was placed on a glass slide the patch had a curious white, shiny, satiny appearance—and it resolved itself under the microscope into a close felt or tangle of most delicate beaded filaments of endless length, exactly resembling those found in the fresh urine. The newly grown terminal portions of a filament were a little slenderer than the older portions, and the component molecules were more perfectly spherical. In the older portions the molecules had a squarer contour—as if from mutual compression. The filaments did not appear to divide or branch in any degree. They appeared to multiply by throwing off single molecules or short pieces from their growing ends, which lengthened into new chains. I could detect no signs of an enveloping sheath. The separate molecules were evidently tightly hung together on their filaments—and the younger threads could be seen to move and twist with a curious uneasy springy motion as they adjusted themselves under the pressure of the covering-glass. There was, however, no real motility of the filaments. The filaments took the aniline dyes readily and yielded beautiful microscopical objects when mounted in Canada balsam.

This organism must be very rare. Though constantly on the watch for it during the past three years I have not encountered a second example. I scarcely think that the organism had any thing to do with the disease of which the patient died. At any rate I have failed to find it in the urine of other patients suffering from undoubted polypoid growths in the bladder. How did the organism gain access to the bladder? As the patient had been more than once sounded for stone before the organism was detected in the urine, the germs of it might be supposed to have been introduced with the instruments—but it is difficult to reconcile this view with the extraordinary rarity of the organism. Bacteruria after the use of instruments, is an every-day occurrence—the urine in such cases is being constantly subjected to minute microscopic examination by competent observers—and yet this very distinctive organism has only been seen in a single case.[1]

X.—ALBUMEN IN THE URINE.

Albumen is not discoverable, even by the most delicate direct testing, in the perfectly normal urine; but it constitutes the most common and most important of the abnormal ingredients found in disease. Its presence in the urine is due to several different conditions, so that the fact itself yields only a vague information; but when correctly interpreted it furnishes a key to certain grave pathological states which would otherwise remain in great obscurity. The kind of albumen found in morbid urines is *serum-albumen*, and in clinical testing our main object is to determine the absence or presence of this variety of albumen in the urine. Albumen is sometimes associated with *globulin* in certain forms of advanced Bright's disease. Globulin is a congener of albumen, and coexists with it in the serum of the blood. Its presence in quantity in the urine is detected by simply diluting the urine with a large amount of water—if globulin be present, the urine thereupon becomes more or less milky in appearance. This *globulin reaction* depends on the fact that globulin is insoluble in pure water, but is soluble in saline solutions; and when urine containing it is largely diluted with water, the salts which keep this substance in solution are reduced to so attenuated a proportion that they no longer suffice for this purpose, and the globulin is thrown out of solution. An albuminous urine, which becomes milky on dilution with water, has its transparency instantly restored by the addition of a few drops of liquor potassæ or of a mineral acid. In all other respects globulin answers to the same tests as albumen, and its presence in the urine does not interfere with the ordinary processes for albumen testing.[2]

In addition to albumen and globulin, the presence of which, in quantities appreciable by direct testing, must always be regarded as abnormal, the urine also contains, both in health and in disease, not unfrequently, certain other albuminoid substances of which it is necessary to take cognizance. These are *peptone, hemialbumose, mucin,* and the coloring-matters of the blood—*hæmoglobin* and *methæmoglobin.*

Peptone and Hemialbumose.—Minute quantities of peptone appear occasionally in the urine of healthy persons, and cannot be said to have any pathological significance. Hemialbumose seems to be very rarely present in the urine. It was discovered in a case of osteomalacia by Dr. Bence Jones,[1] and named by him hydrated deutoxide of albumen. It was subsequently found by Kühne in a similar case, and named hemialbumose. Its chemical reactions are still imperfectly understood. It is precipitated by nitric acid in the cold, but the precipitate is redissolved with excess of the acid on heating. It is not precipitated by boiling. Picric acid throws it down. It is considered as identical with the pro-pepton of Schmidt-Mülheim and with the a-pepton of Meissner, and is one of the transitional phases in the peptic and tryptic digestion of albumen.[2] Peptone in minute quantities seems to be often present in the urine—and occasionally in large quantities, constituting a condition which has been named "peptonuria"—about which, however, very little is known. Peptone is thrown down by picric acid, and by acidulated solution of common salt, but is not precipitated by nitric acid nor by boiling.

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[1] I sometimes think it possible that the parasite is of exotic origin, and that the patient brought it home with him from South America or India. If so, similar cases must be looked for, not in Europe, but in tropical regions.

[2] Globulin may more accurately be detected by saturating the urine with sulphate of magnesia, which precipitates globulin from its solutions.

Mucin.—Traces of mucin in a state of solution seem to be present in all urines, both healthy and morbid; but not unfrequently mucin is present in considerable quantities in the urine of patients suffering from all kinds both of grave and trivial disorders. Further researches are required to indicate its real clinical significance. In searching for minute traces of albumen in the urine, the presence of mucin occasions more frequent embarrassment than any other substance. Mucin is precipitated by the organic acids; and the precipitate is not redissolved by the addition of these acids in excess, nor by boiling. It is not thrown down by the strong mineral acids, but when these are largely diluted, they produce the same effect as the organic acids. The best way of detecting mucin is by means of a saturated solution of citric acid. If such a solution be added to urine in a test-tube, in the same way as in the contact method of applying the nitric acid test for albumen—that is to say, if it be allowed to trickle along the sides of the tube until it forms a distinct layer below the column of urine—there will gradually appear, if mucin be present, an opalescent zone immediately above the layer of acid. Acetic and lactic acids are less appropriate for eliciting the mucin reaction than the strong citric acid solution, because, owing to their less specific gravity, they do not so readily sink to the bottom of the tube, and form a distinct layer below the urine. But if acetic acid be mixed with one-third of its bulk of glycerine, it acquires the due density, and answers perfectly as a mucin test. Sometimes mucin is so abundantly present that the free addition of acetic acid, without any precautions, produces a marked milkiness in the urine.

Hæmoglobin and Methæmoglobin.—These bodies constitute the coloring-matters of the blood, and their presence in urine is recognized by the blood-color which they impart to the secretion. (See Hæmoglobinuria.)

QUALITATIVE TESTING FOR ALBUMEN.—The best tests for albumen are coagulation by boiling, and nitric acid; in doubtful cases the two tests should be used in succession.

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[1] Bence Jones, Animal Chemistry, p. 109.

[2] *See* Salkowski, Die Lehre vom Harn., p. 210; also Dr. Gowers's case, Lancet, 1878, ii. p. 3.

Boiling.—If a urine possessing its usual acid reaction be boiled in a test-tube, it becomes turbid if it contain albumen; and this turbidity is not removed by the addition of an acid. When the urine is turbid from deposition of amorphous urates, boiling alone is a complete—and the best—test for albumen. The deposition of the urates is sufficient evidence that the urine is frankly acid; when such a urine is heated, the urates are speedily dissolved and the urine becomes transparent, but as the temperature approaches the boiling point the urine again becomes turbid if it contain albumen.

In using the boiling test it is of the first importance to attend to the due acidulation of the urine. For if the urine be alkaline any albumen it may contain is not coagulated on boiling. Again, if the urine be alkaline, or neutral, or only slightly acid, it may become turbid on boiling from precipitation of the earthy phosphates. Turbidity from this cause is distinguished from that produced by albumen by the addition of a drop or two of acetic or nitric acid. This immediately dissolves a phosphatic precipitate, but has no effect on albumen.

To avoid these sources of fallacy, the best way is to acidulate the urine before boiling with acetic acid. Care must, however, be taken not to add too much nor too little acid, otherwise the delicacy of the test is much impaired. The following procedure may be relied on to yield trustworthy results. A test-tube is charged with about three fluid-drachms (10 c.c.) of urine. To this is added a single drop of acetic acid. The upper half of the column is then heated to ebullition. If albumen be present, the upper boiled portion of the column will show opalescence, in contrast with the lower half, which remains unchanged. If the urine be alkaline, it should be carefully neutralized by adding successive drops of acetic acid, until the litmus paper shows a distinct, but slight, acidity, and then the final single drop of acid is added before boiling. Even if the urine possesses its natural acidity it is better to add a drop of acid if you want to bring out the maximum sensitiveness of the boiling test. When performed with these precautions the boiling test is the most sensitive and the most reliable of all albumen tests.

Nitric Acid.—Nitric acid is an extremely delicate test for albumen; and it is the first test to use in all cases except when the urine is turbid from urates. The best manner of applying it is to fill a test-tube to the depth of about an inch; then, inclining the tube, to pour in strong nitric acid in such a manner that it may trickle down along the side of the tube to the bottom, and form a stratum some quarter of an inch thick below the urine. Added in this manner there is scarcely any mingling of the two fluids, and if albumen be present, three strata or layers will be observed: one, perfectly colorless, of nitric acid at the bottom; immediately above this an opalescent zone of coagulated albumen; and atop the unaltered urine. If there be only a trace of albumen, twenty or thirty minutes elapse before the opalescent zone becomes visible.

This mode of testing for albumen, whereby the reagent is so introduced that it forms a distinct and separate layer either above or below the urine, has been conveniently termed by Dr. Oliver the *contact* method.

Nitric acid applied by the contact method is the simplest and least troublesome means of detecting albumen in the urine. The reaction of the urine does not interfere with its operation. Only one caution is necessary. In concentrated urines, and especially febrile urines, the addition of the acid is apt to precipitate the amorphous urates, and thus to occasion a turbidity which might be mistaken for albumen. The two conditions are however easily distinguished by observing the level at which the cloudiness begins, and the direction in which it spreads. Albumen begins to coagulate immediately above the stratum of acid, and the turbidity spreads upwards; but the urates first appear at or near the surface of the urine, and the opacity spreads downwards. Heat also readily resolves the doubt; for the urates speedily disappear when the urine is warmed, but turbidity from albumen is not affected by heat.

The urine of patients who are taking cubebs and copaiba is commonly somewhat opalescent, and nitric acid, in the cold, sometimes (not always) increases the opalescence. The sense of smell immediately directs attention to the presence of these drugs, and heat diminishes the opalescence and prevents any turbidity with nitric acid.

In urines which are over-rich in urea, nitric acid, in the cold, causes a slow precipitation of a crystalline mass of nitrate of urea, which, however, is so different in appearance from coagulated albumen that it can scarcely be mistaken for it.

It is further to be remarked, that if the manner above described of testing for albumen with nitric acid be not followed, two notable fallacies may be encountered. On the one hand (as was pointed out by Bence Jones), if the urine be acidified with a small quantity, a drop or so, of nitric acid, the albumen may not be coagulated at all; and on the other hand, if a large quantity of acid (an equal volume) be suddenly added to, and mixed with the urine, the mixture remains perfectly clear, even though it be highly albuminous.

Other Tests for Albumen.—Several other substances, besides heat and nitric acid, precipitate albumen; namely, alcohol, tannin, carbolic acid, chromic acid, acidulated brine,[1] metaphosphoric acid,[2] ferrocyanide of potassium,[3] saturated solution of picric acid,[4] tungstate of soda,[5] potassio-mercuric iodide,[6] and certain other metallic salts. Some of these have been recently strongly recommended as tests for albumen in the urine; and it has been claimed on their behalf that they are superior in delicacy to heat and nitric acid.

Those which have attracted the most attention, and which I have especially subjected to examination, are acidulated brine, tungstate of soda, mercuric iodide, and ferrocyanide of potassium. The common defect of these tests is that they not unfrequently give a reaction with normal urines, or with morbid urines, which do not contain serum-albumen. I believe that the most frequent source of fallacy in their use is not peptone nor hemialbumose, but mucin. They all throw down mucin in a manner indistinguishable (without further testing) from albumen. When the contact method is followed, they yield, if the urine contain mucin, an opalescent zone at the junction of the two fluids, and they yield exactly the same reaction if the urine contain albumen. Now the use of nitric acid avoids this fallacy. The opalescent zone produced at the line of contact by nitric acid is albumen (or globulin), and nothing else. If mucin be present, there is slowly produced a haze at the middle and upper parts of the column of urine, and not at the line of contact. Sometimes the mucin-haze, with very careful addition of the acid, forms a fairly distinct ring or zone about midway between the contact line and the top of the column of urine; and now and then a urine is encountered in which a double zone is witnessed, one consisting of albumen immediately above the line of junction of the two fluids, and another higher up composed of mucin. This behavior of mucin with nitric acid is easily understood when it is remembered that while mucin is not thrown down by strong nitric acid, it is thrown down by that acid in a highly diluted state.

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[1] Proposed by myself. This test is composed of a saturated solution of common salt acidulated with one per cent. of strong hydrochloric acid. See a paper by the author in The Lancet, 1882, ii. p. 823.

[2] Metaphosphoric acid is a very sensitive test for albumen—but its solutions change slowly into orthophosphoric acid and then cease to coagulate albumen. This is a fatal objection to its clinical use.

[3] Suggested by Dr. Pavy. The objection to this test is that it throws down mucin owing to the strong acidulation with acetic acid which is required to bring out its action.

[4] First proposed by Galippe, and recently strongly advocated by Dr. George Johnson (The Lancet, 1882, ii. p. 737, and Albumen and Sugar Testing, Lond. 1884). Applied by the contact method this is a very delicate test for albumen—but inasmuch as it throws down mucin and peptone in a manner quite indistinguishable from albumen (except by further control testing—by heat or nitric acid), its use involves additional time and trouble. Picric acid also gives a precipitate in the urine of persons taking large doses of quinine.

[5] This test was introduced by Dr. Oliver (On Bedside Urine Testing, Lond., 1883). It is prepared by mixing together equal parts of the saturated solutions of tungstate of soda (one in four), and of citric acid (ten in six), and of water. It is applied by the contact method and is of extreme delicacy—but it is fatally vitiated as a clinical test by the fact that it gives a reaction more or less pronounced with most urines both healthy and morbid.

[6] Proposed by Ch. Tanret of Paris. It is composed of 2.70 parts of bichloride of mercury and 6.64 parts of iodide of potassium dissolved in 100 parts of water. In applying this test the urine requires to be strongly acidulated with acetic or citric acid. This test is of the most extreme sensitiveness, but it is wholly unsuitable for urine testing for the same reason as the foregoing, namely, that it gives a slight reaction with nearly all urines.

In regard to highly or even very moderately albuminous urines, all these tests give an unmistakable reaction; but when we are in search of minute traces of albumen they fail us, simply because their reactions do not distinguish between the presence of albumen and the presence of other proteids which have either no morbid significance or have a significance wholly different from that of albumen. It is for this reason that I have been constrained to abandon the use of acidulated brine. I found it so often necessary, when minute traces of albumen were in question, to control the indications with heat and nitric acid, that it became evident that it cost less time and trouble to resort at once to the more reliable tests.

It is, no doubt, desirable that we should possess a test for albumen somewhat more sensitive than nitric acid; but it is a condition, *sine qua non*, that such a test shall be equally reliable, and this condition is not fulfilled by any of the tests hitherto introduced. They all, without exception, give a reaction with something that is not serum-albumen, and are, therefore, untrustworthy, and apt to lead to serious misapprehension. The following table brings into strong light the necessity of abiding by the old tests. It gives the results of the examination of the urine of thirty-one perfectly healthy men, most of them students and candidates for insurance. None of these urines gave the slightest reaction by careful testing with heat and nitric acid:

	Reaction.	No reaction.
Heat	0	31
Nitric acid	0	31
Acidulated brine	11	20
Picric acid	14	17
Tungstate test	28	3
Mercuric iodide	26	5

It may be objected that the failure of heat and nitric acid to give a reaction with these urines arose from a want of sufficient delicacy; but this was not really the case. I have shown elsewhere[1] that the boiling test, when attention is paid to the due acidulation of the urine, is superior in sensitiveness to any of the new tests.

THE QUANTITATIVE ESTIMATION of albumen in urine is a matter of considerable practical importance, and various plans of attaining this object have been devised.

For precise determinations the plan usually followed is to bring a measured quantity of urine to a slightly acid condition; boil: throw on a weighed filter; wash; dry at 212°; and weigh. This proceeding demands a good deal of time. The filtering is sometimes impossible; and the results obtained are only moderately accurate with every care.

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[1] Glasgow Medical Journal, 1884.

For a rough-and-ready, but useful, method, there is none superior to boiling the urine in a test-tube with a drop or two of acetic acid. The albumen coagulates in flakes, and presently sinks to the bottom, forming a layer of various thickness. The proportion of albumen is judged of by the depth of this layer as compared to the height of the column of urine in the tube. This proportion may be expressed in numbers, as $\frac{1}{2}$, $\frac{1}{4}$, $\frac{1}{1}$, and so forth. If the quantity of albumen be too small to form a layer of appreciable depth, the proportion is expressed more loosely, as a "cloudiness" or an "opalescence." The varying density of albuminous urines, and the varying size of the flakes into which albumen coagulates, affect the rapidity and completeness of the subsidence and therefore the depth of the coagulated layer, so that only approximate results can be expected from this method.

Becquerel ingeniously turned to account the property of albumen to deviate the plane of polarization to the left; and constructed an instrument on a similar plan to the optical saccharimeter, by which the deviation could be measured, and the percentage of albumen calculated therefrom. It would appear, however, that this instrument, on Becquerel's own showing, is only capable of very limited clinical application. When the quantity of albumen is considerable it gives very exact indications; but the deviation is too slight for exact estimation in moderately and feebly albuminous urines; it is therefore useless for the bulk of albuminous urines.[1]

Boedecker has proposed a volumetrical method, founded on the property of ferrocyanide of potassium to form an insoluble compound of fixed composition with albumen. Vogel states that he has found this method inaccurate.[2]

Minimetric Method or Dilution Method.—In 1876 I proposed a mode of estimating albumen in urine, which I think may prove useful in clinical work.[3] The principle of the method is easily understood.

When an albuminous urine is progressively diluted with water, and tested from time to time with nitric acid, the opacity induced by the test becomes gradually fainter and fainter, until at length it ceases to be visible. This point is reached when the urine contains less than about 0.0014 per cent. of albumen. The more albumen the urine contains, the more dilution, of course, it will require to reach the vanishing point of the reaction; and if we could fix this point with accuracy, we should have a simple method of estimating albumen in urine. The urine could be diluted until it ceased to react with nitric acid, and the amount of dilution required to reach this point would furnish a measure of the proportion of albumen.

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[1] *See* a clinical lecture by Becquerel, Clinique Européene, 1859.

[2] Boedecker's method is described in Henle and Pfeufer's Zeitsch., 1859, p. 321.

[3] For a fuller account of this method the reader is referred to a paper, by the author, read before the Medico-Chirurgical Society, Feb. 22, 1876.

But it is not possible to fix this point with accuracy. The opacity produced by the acid fades away so gradually with increasing additions of water, that it is impracticable to decide within many degrees the point at which the reaction ceases to be appreciable. And not only so, but the development of the reaction becomes more and more retarded as the dilution proceeds, until at length it only becomes visible after the lapse of several minutes.

To overcome this difficulty, it was found necessary to fix on some arbitrary point or line which would serve as a practicable zero to the scale. After many trials, it was found most convenient to draw the line at a reaction coming into sight midway between half and three-quarters of a minute after the contact of the acid—that is, to dilute the urine until it gives no reaction for thirty seconds after the addition of the acid, but shows a distinct opalescence at the forty-fifth second. The exact point to be aimed at is a reaction coming doubtfully into view between the thirty-fifth and fortieth second, and appearing still very dim, but unmistakable at the forty-fifth second. It was found possible, after a little practice, to strike this point with sufficient exactness to serve as a practicable zero to the scale.

Each dilution with an equal volume is counted as one degree on the scale, and these degrees may be conveniently termed "degrees of albumen." Thus, a urine requiring dilution with forty times its bulk of water to reach the zero reaction, may be described as possessing forty degrees of albumen—a urine requiring three hundred similar dilutions as possessing 300 degrees of albumen, and so forth.

The difficulty of the method is to hit correctly the zero reaction. When this point is approached, a little more or a little less dilution makes but a slight difference in the time at which the reaction appears. In order therefore to obtain exact results, it is necessary to conduct the testing with rigid uniformity. The test-tube employed should have an interior diameter of $\frac{5}{8}$ of an inch (15 millimetres); the acid must be added in the right way, and at the right moment. The operation, too, should be performed by daylight, or, if by gaslight, an addition of about five per cent. must be made to the results. The proceeding adopted is as follows: The urine is first tested in the usual way with nitric acid, so as to get a rough idea of the quantity of albumen contained in it, and of the degree of dilution likely to be required to reach the zero. The watch is placed on the table before the operator. A fluid-drachm of the urine is then measured off, and introduced into a graduated pint measure, and water is added up to a few or many ounces, according to the degree of dilution likely to be required to approach the zero reaction. The test-tube is then filled to the depth of about an inch with the diluted urine and held widely inclined from the perpendicular. The eye is now directed to the watch, and the acid is added in such a manner that it runs along the lower side of the tube to the bottom and forms a distinct layer, about a quarter of an inch deep, below the diluted urine. The acid must be added exactly on one of the quarter-minute strokes. This is the most critical step in the proceeding, and it should be performed in the following manner: A pointed glass tube or pipette is dipped to the depth of a couple of inches into the acid and covered with the forefinger. The pipette thus guarded is then passed into the test-tube to within half an inch of the level of the diluted urine, and at the right moment the finger is removed and the charge of acid delivered. As soon as the acid is added, the test-tube is held up to the light against some dark background (such as a black sleeve, a book bound in black cloth, or a dark corner of the room), and as soon as the faintest opalescence is perceived above the level of the acid, the time of its appearing is noted. If this appear at or before thirty seconds after the contact of the acid, more water is added, and the testing repeated as before. Thus, by successive additions of water, and repeated testings, a close approximation to the zero reaction is obtained. A fresh dilution is then prepared, and, guided by the previous trials, two or three testings with different dilutions are generally sufficient to indicate with exactness the dilution which produces an opalescence between the thirty-fifth and forty-fifth second after the addition of the acid.

If too much water is added in the first instance, the reaction does not appear till after the forty-fifth second. In this case the operation must be recommenced with less water, and proceeded with as in the first case.

When the zero reaction is determined, the degree of dilution required to produce it is noted, and expressed in multiples of the unit-volume of urine employed. Thus, if a fluid-drachm was the unit-volume of urine employed, and the zero reaction was obtained when dilution was carried up to fifteen ounces (120 drachms), the urine is recorded as having 120 degrees of albumen.

If the urine is feebly albuminous—indicating less than 20 degrees of albumen—the fluid-ounce should be substituted for the fluid-drachm as the unit-volume. On the other hand, if the urine indicate more than 160 degrees of albumen, the unit-volume should be half a drachm—or, still better, the urine should be previously diluted with water in the proportion of 1 in 2 or 1 in 4, and the result afterwards multiplied by 2 or 4 as the case may be.

The actual value in weight of albumen of each degree on the dilution scale was found, by careful comparative experiments with the weighing method, to correspond to 0.0034 per cent., or 0.0148 grain per fluid-ounce of the British Pharmacopœia. These data supply an easy means of calculating the quantity of albumen per ounce, and also the daily loss of albumen. Suppose that 40 ounces of urine were voided in the twenty-four hours, and that a sample of this urine showed 150 degrees of albumen by the dilution method, then:

$$0.0148 \times 150 = 2.22 \text{ and } 2.22 \times 40 = 88.8.$$

The urine contained 2.22 grains of albumen per ounce, and the daily loss was 88.8 grains.

The time required for the estimation of albumen in urine by this method is from ten to twenty minutes.

Clinical Significance of Albumen in the Urine.—In considering this subject all those cases are, of course, excluded in which the occurrence of albumen is only incidental to the presence of some other fluid in the urine, such as blood or pus.

The pathological states in which albumen appears constantly or occasionally in the urine may be arranged in the following groups:

1. Acute and chronic Bright's disease of the kidneys.
2. Pregnancy and the puerperal state.
3. Febrile and inflammatory diseases (zymotic diseases, such as scarlet fever, measles, smallpox, typhoid, cholera, yellow fever, ague, diphtheria, etc.; inflammatory diseases, such as pneumonia, peritonitis, traumatic fever, acute articular rheumatism, etc.).
4. Impediments to the circulation of the blood (emphysema, heart disease, abdominal tumors, cirrhosis, etc.).
5. A hydræmic and dissolved state of the blood and atony of the tissues (purpura, scurvy, pyæmia, hospital gangrene); also hæmoglobinuria.
6. Saturnine intoxication.
7. Functional disorders (albuminuria of adolescents, physiological albuminuria).
8. Nervous disturbance (neurotic albuminuria).

In the first group albuminuria is dependent on structural changes in the kidneys (*see* Bright's Disease).

In the second group albuminuria is sometimes associated with structural changes, and sometimes not (*see* Connection of Bright's Disease and Pregnancy).

In all febrile and inflammatory complaints a trace of albumen is frequently found in the urine; it usually amounts to no more than a trace, and disappears on defervescence; sometimes in pneumonia it is not inconsiderable. As intercurrent febrile attacks are common in the course of most chronic complaints, temporary albuminuria has been noted in a great multitude of different diseases. This remark applies especially to chronic tuberculosis, cancer, caries, and necrosis; and albuminuria under such a condition is to be carefully distinguished from the cases in which genuine Bright's disease coexists with those complaints. In a zymotic disease there is a double pathological state, namely, pyrexia and the operation of a specific poison; and albumen may appear in the urine either as an incident of the febrile state, when it is comparatively unimportant, or as an indication of serious structural changes in the kidneys, which constitute a grave sequela of the disease.

Albuminuria connected with impediments to the circulation of the blood is considered under Congestion of the Kidney.

In a dissolved or putrid state of the blood, albumen appears in the urine without being connected with organic changes in the kidney; it is associated with the escape of the coloring matter of the blood (*see* Hæmoglobinuria).

Saturnine Albuminuria.—The occurrence of albumen in the urine of persons poisoned with lead, although repeatedly observed, was not regarded as anything more than a coincidence until Ollivier demonstrated, by experiments on animals and clinical observations, the existence of a causal connection between them. Ollivier[1] found that dogs, rabbits, and guinea-pigs, when poisoned with repeated doses of carbonate of lead, invariably passed an albuminous urine, and that their kidneys exhibited signs of incipient organic disease. He also collected 15 examples of albuminuria in persons poisoned with lead. Seven of these had temporary albuminuria; in three, the albuminuria persisted during the continuance of the saturnine symptoms; and in four, genuine Bright's disease had been produced. In addition to these observations, he examined the urine of 37 persons affected with diverse manifestations of lead-poisoning in the Hôpital de la Charité: of these, nine had albuminous urine. These observations have been confirmed by Lancereaux[2] and Danjoy.[3] Ollivier found that both the urine and the kidneys in those cases contained traces of lead. He inferred that the existence of lead in the kidneys induced an organic lesion of these organs, and that the albuminuria was the consequence of that lesion.

This has been proved experimentally by Charcot and Gombault[1], who fed animals on lead salts, and so induced nephritis. Numerous observations on the human subject have clearly established the relation between lead poisoning and the granular kidney of Bright's disease (*see* Dr. Dickinson on "Albuminuria," p. 382, and Wagner on "Die Bleischrumpfniere," in "Ziemssen's Cyclopæd.," 3d edition, vol. ix. p. 291).

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[1] Archives Générales, 1863, ii. pp. 530 and 709.

[2] Union Médicale, 1863, and Bulletins de la Société médicale d'Emulation, nouvelle série, t. i. p. 182, 1864.

[3] Archives Générales, 1864, i. p. 402.

Functional Albuminuria.—The excessive use of a diet composed exclusively or chiefly of albuminous matters, such as eggs, has been shown by Barreswil, Brown-Séquard and others to cause the urine to become slightly albuminous.

Slight and temporary albuminuria appears to occur occasionally from very slight disorders. Beneke, when suffering from dyspepsia, noticed albumen in his own urine four times in as many weeks. Similar observations have been made by others (Parkes).

The most important cases, however, which may be classed under this heading, are those of the so-called *albuminuria of adolescents*, *transient*, or *physiological albuminuria*. The urine of young persons, usually at about the time of puberty, not unfrequently contains a small amount of albumen, without any other serious symptom showing itself. In this country special attention has been called to this form of albuminuria by Moxon,[2] Rooke,[3] Dukes,[4] and Saundby,[5] while on the Continent the most important observations have been those of Leube[6], Edlefsen,[7] Fürbringer,[8] and Runeberg[9]. In most cases the subjects of the affection show general want of tone, lassitude or anæmia, and often disorders of digestion may be noted. But in many cases the general health is quite perfect. The amount of albumen is usually only slight, but occasionally may be considerable. It is a marked feature of nearly all such cases, however, that the appearance of the albumen is intermittent. Thus, the urine passed before breakfast is usually normal, while albumen makes its appearance later on in the day. Occasionally, too, only the urine passed after meals contains albumen. An excess of uric acid or of oxalates, and sometimes a few hyaline casts, may be found. Muscular exertion has a very marked effect in inducing this form of albuminuria. Leube examined the urine of 119 soldiers after a long march, and found albumen in the urine of 19, or 16 per cent. Dr. Dukes has also remarked in schoolboys the effect of muscular exertion in producing albuminuria; while rest in bed, as shown by Dr. Rooke, frequently removes the condition in a short time.

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[1] Archiv. de Physiol., 1881, p. 126.

[2] Guy's Hosp, Reports, xxiii. p. 233.

[3] Brit. Med. Journ., 1878, ii. p. 596.

[4] Idem, 1878, ii. p. 794.

[5] Idem, 1879, i. p 699.

[6] Virch. Arch., 72, p. 145.

[7] Berlin. Klin. Wochenschr., Sept. 12, 1879.

[8] Zeitsch. f. Klin. Medic., 1879, i. p. 340.

[9] Virch. Arch., 80, p. 175.

Usually the condition is not of long duration, but exceptions to this rule are sometimes met with.

The causes of functional albuminuria are by no means understood, and probably may not be the same in all cases. Some cases may possibly be regarded as food-albuminuria. Sir Wm. Gull has suggested that the condition is due to atony of the vessels and nerves, and Bamberger also believes it due to vasomotor change in the kidney, which may cause slowing of the circulation in the glomeruli. This appears to be the most probable explanation for most cases.

The prognosis in functional albuminuria is favorable—although Dr. G. Johnson[1] and Dr. Dukes are inclined to think that sometimes the condition may end in Bright's disease.

The functional character of the disorder may generally be recognized from the intermittent occurrence of the albumen, from the normal density and coloration of the urine, from the absence of other accompaniments of Bright's disease, such as high arterial tension and hypertrophy of the heart, and from the progress of the case.

Neurotic Albuminuria.—Bernard found that irritation of the renal nerves, or of a certain spot in the floor of the fourth ventricle (higher up than the diabetic puncture) caused albumen to appear in the urine of animals. Temporary or intermittent albuminuria is sometimes encountered clinically under circumstances of disturbed innervation, without structural changes in the kidneys. Dr. G. Johnson[2] has pointed out that transient albuminuria occasionally follows cold bathing, and Dr. Laycock[3] has seen a similar result in the shivering period of various ague-like attacks. In vascular bronchocele with exophthalmos, Dr. Begbie[4] has repeatedly observed long-continued intermitting albuminuria. In these last cases the albumen appears during and after digestion (especially after breakfast), and disappears during the periods of fasting. Some cases of this class have an evident affinity with paroxysmal hæmoglobinuria.

Albuminuria is sometimes noticed after epileptic attacks, but the records of the frequency of its occurrence show considerable differences. In certain other disturbances of the nervous system albuminuria is noticed, as in cerebral hemorrhage, cerebral concussion, tetanus, and delirium tremens. (*See* Wagner in "Ziemssen's Cyclop.," 3d. edit., vol. ix. p. 27.)

When albumen is found in urine, the important point to decide is, whether it indicates the existence of organic disease of the kidneys or not. This question, in any individual case, must be considered chiefly in connection with the three following points jointly, namely:

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[1] Brit. Med. Journ., 1879, ii. p. 928.

[2] Ibid., 1873, ii. p. 664.

[3] Dublin Journ of Med. Sci., July, 1874.

[4] Edin. Med. Journ., April,1874.

1. The temporary or persistent duration of the albuminuria.

2. The quantity of the albumen; and the occurrence and character of a deposit of renal derivatives.

3. The presence or absence of any disease outside the kidneys which will account for the albuminuria.

1. It has already been mentioned that functional albuminuria is usually only temporary. A persistent duration of albuminuria, on the other hand, is very suspicious of organic disease of the kidneys. The importance of distinguishing between temporary and permanent albuminuria was insisted upon by Dr. Parkes, and observations relating to this subject will be found in his treatise "On the Composition of the Urine," p. 186. It must be remembered that in the granular kidney of Bright's disease the albuminuria is often intermittent.

2. The greater the quantity of albumen, the more likely is the existence of renal disease; and a "large" quantity of albumen ($\frac{1}{4}$ and upwards) is rarely found, except in undoubted acute or chronic Bright's disease. It is necessary, however, in considering the amount of albumen, not only to have regard to the proportion in a particular specimen examined, but also to the total quantity in the twenty-four hours. This may be surmised by the density of the urine—low density indicating that the quantity of urine passed in twenty-four hours is large, and high density the contrary—but judged more accurately by ascertaining what is the actual flow of urine in twenty-four hours. A urine may be only slightly albuminous, but if it be of low density (under 1012) and the daily quantity between three and four pints, the total loss of albumen will be very considerable, and the existence of renal disease strongly indicated. Indeed, of all urines there is none more surely indicative of Bright's disease than a pale, dilute, abundant urine which is, at the same time, more or less albuminous. On the other hand, as a rule, with very few exceptions, when the urine is only slightly albuminous, and at the same time dense and high colored, Bright's disease is not present, and the albuminuria is owing either to pyrexia or to some impediment in the circulation of the blood.

The kinds of deposit which indicate most strongly the existence of organic renal disease are, (*a*) very abundant ones, containing casts and much renal epithelium; (*b*) those containing numerous casts and cells in a state of fatty degeneration. The least indicative of primary renal disease of serious import, are, blood casts, and very transparent casts in scanty numbers.

3. When the urine is found permanently albuminous, and there exists neither pyrexia nor thoracic disease, nor other recognizable condition which can account for the albumen, the

inference is almost irresistible that there exists a primary organic disease of the kidneys. The association of other symptoms will usually confirm this diagnosis. (*See* Bright's Disease.)

It has been stated that it is possible to distinguish secondary and functional albuminuria from albuminuria depending on disease of the kidneys, by the fact that certain odorous and pigmentary substances when taken internally make their appearance in the urine in the former case (as in health) but not in the latter. The observations of Dr. Dyce Duckworth do not support this conclusion. He found that iodine, santonine, turpentine, and oil of juniper passed through the kidneys, and appeared in the urine, of persons affected with undoubted disease of the kidneys. Some exceptional cases were, however, encountered.[1]

PATHOLOGY OF ALBUMINURIA.

It would be out of place in a practical treatise to discuss fully the various theories which have been advanced in recent years to explain the presence of albumen in morbid urine. The subject, however, is of so much interest, and has attracted so much attention, that a short review may fitly be here inserted, giving in outline the views of pathologists on this vexed question.

As a preliminary inquiry it must be considered whether albuminuria is an exclusively morbid phenomena, or whether the urine may in the normal condition contain a certain quantity of albumen—a quantity so minute as to escape detection by the ordinary tests. The appearance of albumen in functional albuminuria would by the last view be simply an exaggeration of a physiological condition. We should thus have a sensible and insensible albuminuria, analogous to sensible and insensible perspiration. The systematic testing of the urine, especially in candidates for life insurance, has brought to light the frequency with which albuminuria occurs when organic disease of the kidneys, or, indeed, of any other organ, cannot be suspected (*see* above—Functional Albuminuria). It is now certain that a sensible albuminuria is of much commoner occurrence in healthy persons than has been hitherto supposed. Some of the constituents of the urine which were once considered exclusively morbid, have, by the use of finer methods of testing, been found to exist in minute quantities under perfectly normal conditions. For example, it has been shown that minute traces of sugar exist in normal urine—and analogy would suggest that albumen also may in the future be shown to be an ingredient of the normal secretion of the kidney, although, perhaps, present only at certain periods of the day. Such a view is already held by some physicians (*see* Senator, "Die Albuminurie in gesunden und kranken zustande," Berlin, 1882). The majority of pathologists, however, still hold that every appearance of albumen in the urine is morbid, although it may not necessarily point to a diseased condition of the kidneys.

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[1] St. Bart. Reports, vol. iii. p. 215.

How comes it to pass that while so many other substances are removed from the blood by the kidneys, the albumen is retained? To this question mainly two answers have been given. The first is that of Von Wittich,[1] who believed that in the glomeruli albumen really was removed from the blood, but that it was afterwards reabsorbed in the tubules and served to nourish their epithelium. This view has met with many opponents, and direct contradiction has been given to it, by the experiments mentioned below, which showed no coagulated albumen in the glomerular capsule, when the kidney was boiled, or when treated with alcohol or osmic acid immediately after removal from the body. The view to which authorities are now most inclined is that advocated by Heidenhain.[2] He accepts the opinion of Bowman that the urinary salts and water are removed in the glomeruli, and that the specific urinary constituents, such as urea, are secreted by the epithelium of the urinary tubules. The separation of the water and salts, however, is not a mere filtration as formerly supposed. The glomerular tuft is covered by a layer of flat epithelium, which, according to Heidenhain, is an active agent in removing the water and salts from the blood, but keeps back the albumen, and prevents it reaching the urine at all. We have, in fact, a true secretion, the glomerular epithelium acting towards the water and salts, as the tubular epithelium towards the urea, uric acid, etc. Moreover, any lesion of this epithelial layer, or a deficiency in its supply of nutrition, would cause it to lose part of its function and permit the passage of albumen into the urine.

Support is lent to this opinion by a consideration of the part of the kidney into which albumen is exuded when present in the urine. Varied experiments have now placed it beyond doubt that the albumen is, for the greater part at least, poured out in the glomerular capsule. The experiments have been conducted in two ways. The kidneys of amphibia have a double blood supply. The glomeruli are supplied from the renal artery, while the tubules are supplied by a sort of portal vein, which springs from the veins of the lower extremity. Nussbaum[3] showed that by ligaturing the renal artery in frogs he could shut off the blood supply from the glomeruli, while leaving that of the tubules intact. He then produced an artificial albuminuria in frogs by the injection of egg-albumen into the anterior abdominal vein; but he found that the excretion of albumen immediately stopped when he ligatured the renal artery, although other substances were still secreted by the intact epithelium of the tubules. It could, therefore, be concluded that in this case albumen was only secreted by the glomeruli. Another method employed was that of coagulating the albumen *in situ*, by treating the kidney with various coagulating agents immediately after removal from the body. For this purpose Cornil[4] used osmic acid, Ribbert[5] strong alcohol, and Posner[6] boiled the kidney for a short time. When sections of the kidney treated in this way were examined, a mass of coagulated albumen could be seen not only in the tubes, but in the space between the glomerular tuft and its capsule. Hence, it must be inferred that usually the albumen is secreted by the glomeruli. Senator finds an exception to this in the case of venous congestion, where he believes that the albumen is first removed in the tubules, since the increase of pressure in the venous system will be felt here before it reaches the glomeruli.

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[1] Virch. Arch., x. p. 325.

[2] Hermann's Handb. der Physiologie, Bd. v., part i.

[3] Pflüger's Arch., xvii. p. 580.

[4] Journ. de l'Anat., 1879.

[5] Clblatt. f. Med. Wissen., 1879.

[6] Virch. Arch., 79, p. 311.

The general conditions which give rise to albuminuria may be classified as follows:

I.—Alterations in the Composition of the Blood.

Mention has already been made of the classic experiments of Bernard, Lehmann, and Stokvis, by which it was shown that certain *foreign* albumens, when present in the blood, passed over into the urine unchanged. Such albumens are egg-albumen and hæmoglobin, which are more diffusible than serum-albumen; while others, such as syntonin, myosin, and alkali albuminate, being less diffusible, remain in the bloodvessels (Lehmann). The presence in the digestive tract of albuminous bodies in excess, may be followed by their absorption and excretion by the kidneys, in the unchanged state. The late Sir Robert Christison pointed out, in 1839, that the urine of a person who ate much cheese might contain albumen. Bernard, Stokvis, Lehmann, and many others, have also shown that the use of uncooked eggs as an article of diet, or an excessive use of them when cooked, may lead to the presence of egg-albumen in the urine. The question of a "food albuminuria" has been investigated by Parkes, and more recently by Brunton and Power[1] and Sparks and Bruce.[2] They have proved that albumen may occur in the urine after taking food; and if present before food, it is increased in amount during digestion. Such observations, however, do not prove that the albumen absorbed is at once excreted by the kidneys, for changes in the blood circulation are coincident, and they also may lead to disturbance of the renal function. It has been surmised that the albumen found in the urine differs slightly from ordinary serum-albumen. Brunton and Power investigated the coagulating-point of the albumen, and found that the albumen after food did in some cases coagulate at a lower temperature than ordinary serum-albumen, and that the earlier products of pancreatic digestion showed the same phenomenon. Their experiments, however, did not give constant results. Lépine[3] asserts that the albumen passed after food is more diffusible, and more easily transformed into peptone by artificial digestion than that passed during fasting. It is not improbable that special forms of albumen, such as hemialbumose, peptones, and possibly transition stages between serum-albumen and these, may be absorbed from the digestive tract and excreted in the urine. Peptones, it is alleged, may be absorbed from pathological exudations, as in croupous pneumonia, and during the breaking down of purulent formations in various parts of the body. It is asserted that in fevers and certain other conditions, peptones may be formed in the blood itself by a peculiar fermentative process, and may then appear in the urine. (*See* Senator, *loc. cit.* p. 8.)

The excretion of other forms of albumen may, however, lead to the exudation of serum-albumen itself. Lehmann and Stokvis showed that albuminuria did not end with the removal of all the foreign albumen injected into the blood, but that a quantity of serum-albumen was passed for some time. It is, therefore, probable that the passage through the kidney of such foreign albumen may irritate the organ and lead to permanent changes. Semmola[1] believes that the lesion of the kidney in Bright's disease is thus produced by the continued excretion of unassimilated albuminoid matters which accumulate in the blood. His opinion, however, seems to rest on insufficient experimental data.

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[1] St. Bart. Reports, vol. xiii.

[2] Med.-Chir. Trans., vol. lxii.

[3] Revue Mensuelle, 1880, p. 343.

The occurrence of functional albuminuria is by some authors attributed to derangements of digestion (dyspeptic albuminuria). The late Dr. Murchison[2] also advocated the admission of a hepatic albuminuria. The liver, he believed, might be so overworked as to allow a portion of albumen to pass over into the blood in an unassimilated state, to be afterwards removed by the kidneys.

With reference to other constituents, the relation of the composition of the blood to albuminuria has, as yet, been insufficiently worked out. Dr. Newman,[3] however, has shown experimentally that the amount of albumen secreted is increased by an accumulation of urea in the blood.

II.—Alterations in the Circulation of Blood through the Kidneys.

These may consist of changes in the quantity of blood supplied to the kidneys, in the blood pressure, or in the rate of blood flow. Our knowledge of the action of these changes is derived mainly from experimental pathology; but it is extremely difficult, and in some cases impossible, to vary one set of relations apart from the others, and, therefore, the results of the experiments and the deductions drawn from them are somewhat conflicting.

If the renal artery be closed for a few minutes and then reopened, the secretion of urine is stopped for a time, and only gradually reestablished. The urine passed after the reopening of the artery contains a quantity of albumen, which gradually disappears as the flow of urine becomes normal. (Hermann[4] and Overbeck.[5]) A parallel to this experiment is probably seen in the algid stage of cholera. The supply of blood to the kidneys is then interrupted, and the secretion stopped; but in the stage of reaction, if the patient survive, the urine is found to contain albumen, which gradually disappears as convalescence progresses. Hermann also showed that mere narrowing of the renal artery, without absolute occlusion, would cause a similar result. It cannot, however, be concluded that the results are due to anæmia of the kidney. They have been variously referred to lowering of blood pressure, increase of blood pressure from accumulation of corpuscles in the glomerulus during the stasis, to slowing of the blood stream, and to loss of nutrition of the glomerular epithelium.

Similarly, it is a matter of every-day clinical experience that hyperæmia of the kidneys is accompanied by albuminuria. Here, however, we have again the same complication of relations, vascular tension and blood flow being alike altered.

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[1] Revue Mensuelle, 1880, p. 239; also Progrès Médical, 1883.

[2] Diseases of the Liver, 2d edit., p. 573.

[3] Journ. of Anatomy and Phys., vol. xii. p. 608.

[4] Sitzungsb. d. Wien. Acad., 1861.

[5] Idem, 1863.

It has been very generally believed that increase of arterial tension would cause albuminuria. This seemed to be proved clinically by Dr. Mahomed,[1] who showed that the appearance of albumen in the urine as a sequel of scarlet fever is preceded by a period of high vascular tension.

Experimentally, also, the same conclusion is arrived at. Thus, Robinson[2] and Frerichs[3] found that by ligaturing the abdominal aorta below the renal arteries and then removing one kidney, they caused albumen to appear in the urine coming from the remaining kidney. More recently Lépine (*loc. cit.*) has produced rise of blood pressure and consequent albuminuria by injecting a quantity of salt solution into the crural vein of a dog.

An astonishing theory was propounded by Runeberg ("Deutsch. Arch. f. Klin. Medic.," vol. 23), as the result of his experiments on the diffusion of albuminous fluids through animal membranes. He attempted to show that the transudation of albumen was favored by a diminution in the difference of pressure on the two sides of the dialyzing membrane, and hindered by an increase of such difference. Applying his view to the kidney, he asserted that the cause of albuminuria was low vascular pressure, which produced increased permeability of the walls of the Malpighian tufts. Runeberg's results were, however, entirely contradicted by the careful experiments of Dr. Newman (*loc. cit.*), and also by those of Bamberger[4] and Gottwalt.[5] Heidenhain, too, has shown that Runeberg's own figures will not bear the interpretation he himself placed upon them, while Bamberger points out that Runeberg has paid no attention to coincident variations of blood flow. It must, therefore, be accepted that low vascular pressure is not a cause of albuminuria.

It is not safe, however, to assert that increased pressure in the Malpighian capillaries will alone cause albuminuria, for it is impossible entirely to eliminate other influences which may at least assist the high pressure in its work.

There is now a fairly general agreement that slowing of the blood stream plays a very active part in producing albuminuria (Bamberger and Heidenhain). Dr. Mahomed[6] groups the two conditions, increased pressure and retardation of blood flow, as the chief factors in the causation of albuminuria.

The modes in which these various changes in the blood system of the kidney may be brought about, are of course numerous. Increase of blood pressure in the kidney may be caused by general rise of blood pressure, or locally through the vaso-motor nerves. The fact that puncture of the floor of the fourth ventricle may produce albuminuria is probably to be explained by coincident injury to the vaso-motor centre. Bamberger believes that the vaso-motor system may be efficient in producing many cases of functional albuminuria. Weakness of the heart's action may cause slowing of the blood stream in the kidney, and venous obstruction may produce not only slowing of the blood stream, but also increase of pressure in the glomeruli.

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[1] Med. Chir. Trans., vol. 57.

[2] Med. Chir. Trans., vol. 26.

[3] Die Bright'sche Nierenkrank, Braunschweig, 1851.

[4] Wien. Med. Wochensch., 1881.

[5] Zeitsch. f. Physiolog. Chemie., IV. p. 423.

[6] Glasgow Med. Journ., 1884.

III.—Alterations in the Structure of the Kidney.

These are admitted by all to be efficient causes of albuminuria.

The change in structure may affect either the kidney epithelium or the bloodvessels. If it be accepted that the glomerular epithelium in the normal state prevents the albumen exuding from the blood, it is plain that any diseased condition of this epithelium will allow the albumen to pass through. A similar function has been advocated by Senator for the epithelium of the renal tubes. Not only may gross lesions of these epithelial layers cause albuminuria, but it is also considered that simple lesions of nutrition may lead to loss of function. Thus, Cohnheim[1] asserts that every important change of circulation renders the secreting membrane permeable by albumen. The lesions, then, which have been described above, such as narrowing or obliteration of the renal artery, changes in blood pressure, and slowing of the blood stream, would produce their effects by causing disordered nutrition of the epithelium covering the glomeruli. The nutritive change may be temporary, or may lead to permanent changes. The proofs of this view, however, are difficult to obtain, and it must at present be considered as *sub judice*.

Increase of interstitial tissue, as is found in granular kidney, may be accompanied not only by changes in the epithelium, but by hindrance to the blood flow, from compression of the capillaries by the new growth.

The effect of structural changes in the bloodvessels is manifest in the albuminuria which usually accompanies amyloid degeneration of the kidney. Here we have as the initial lesion a degeneration of the walls of the bloodvessels, which renders them more permeable to albumen.

Such are the individual causes of albuminuria, but it will be gathered from what has been said that the causes do not act singly, and however much stress may be laid upon one change in any particular pathological state, that change is almost invariably assisted in its action by others. Thus in the albuminuria of fevers we have structural change, as shown by the cloudy degeneration of the renal epithelium, we have also changes in the blood pressure and in the rate of blood flow, while it is by no means improbable that the albumen of the blood may also undergo modification.

XI.—SUGAR IN URINE.

In 1862 Schunck[2] announced that, when healthy urine was subjected to boiling with acids, it gradually deposited a resinous substance, and acquired the power of reducing the oxide of copper—in other words, that the presence of a substance having the properties of glucose became apparent in it. This important observation probably explains the discrepant conclusions of those who have sought for sugar as a normal constituent of healthy urine. Brücke[1] and Bence Jones were always able to obtain sugar from healthy urine in not inconsiderable quantities. Bence Jones[2] obtained as much as 0.8 to 1.7 grain per pint. If natural urine contain a substance, capable of yielding sugar by a simple decomposition, it is quite possible that the sugar found by these observers was, either partly or wholly, an educt of the analysis, and not a preëxisting constituent of the urine. This much is certain, that healthy urines and the vast majority of morbid urines, do not contain sugar in quantity capable of being detected by ordinary direct testing. At the same time it is probable that minute traces of sugar, as of nearly every other substance dissolved in the blood, exist in the urine. These traces, however, granting them to exist, have no clinical significance whatsoever. When sugar is present in quantity sufficient to interest the medical practitioner, it is detectable with certainty by direct testing; and conversely when direct testing reveals the presence of sugar, it is invariably a grave pathological sign, and not a matter of mere physiological curiosity. In the following observations I have solely in view sugar in these sensible proportions.

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[1] Allgemeine Pathologie, vol. ii. p. 315.

[2] Philosophical Magazine, March, 1862.

TESTS FOR SUGAR IN URINE. (QUALITATIVE TESTING.)—Frequent mistakes are committed in regard to the presence or absence of sugar in urine, not only by physicians and surgeons, but even by professed chemists. More than once, specimens have been brought to me with the statement that an analytic chemist had found a small quantity of sugar, but in which no sugar really existed—certain fallacious appearances, to which I shall presently refer, having been mistaken for genuine evidence. Without proper precautions, sugar testing, like all other testing, is open to fallacies; but with moderate care and observance of a few fixed rules, the detection of sugar is a matter of the most perfect certainty and of exquisite delicacy. Before proceeding to describe the *best* means for this purpose, I will say a word about those tests which are in common use, but which are either unreliable or insufficiently delicate—namely, Moore's test, and the fermentation test.

1. *Boiling with Liquor Potassæ (Moore's Test).*—When urine containing sugar is boiled with an equal bulk of liquor potassæ, the

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[1] Iwanoff has pointed out some fallacies in Brücke's process. He considers that the greater part of the sugar obtained by Brücke did not preëxist in the urine, but was derived from some other constituent (indican) by the reagents employed. Iwanoff concludes that minute traces of sugar do exist frequently, but by no means constantly in healthy urine. (Meissner's Bericht in Henle and Pfeufer's Zeitsch. for 1861, p. 323.) In Bence Jones's process sulphuretted hydrogen was employed instead of oxalic acid; but even with this modification, the urine would be rendered acid, and there would be great probability of sugar being produced from indican during the long process of evaporation of the large quantities (1000 c.c.) of urine used.

[2] Journal of the Chemical Society, 1862, p. 22.

mixture darkens, and eventually assumes a brandy-brown color. From its easy application, this test, as a preliminary step, and for negative evidence, is of great convenience. It has, however, two faults—(*a*) it is wanting in delicacy, and (*b*) it is liable to a notable fallacy. Moore's test does not answer clearly until the proportion of sugar rises to about 0.3 per cent., or one grain and a half to the ounce. By-and-by we shall come to a test *twelve* times more delicate than this.

Again, all high-colored urines of high density become darker when boiled with liquor potassæ, although free from sugar; and albuminous urines, even when not high-colored, darken sensibly under the same treatment. This occurs with perfectly fresh liquor potassæ; but if the test have been kept in ordinary white-glass bottles, it very speedily becomes impregnated with lead, which it attracts from the glass, and this offers an additional source of error. The liquor potassæ kept in the wards of the Royal Infirmary was found largely impregnated with lead, although it had not been in use more than about six weeks. Liquor potassæ thus vitiated, when boiled with certain urines, turns them of a dark porter-brown color. This is something quite different from the slight deepening of the tint which has just been alluded to, and it only occurs in *albuminous* urines, and not even in all of these. In acute Bright's disease, especially when there was blood in the urine, or when the albumen was abundant and associated with free discharge of renal epithelium, the change of color was most intense; and in one such case the existence of sugar had been inferred therefrom, and announced to the patient and his friends, by the medical attendant. Where the proportion of albumen was small, and renal desquamation slight, the lead-tainted liquor potassæ did not produce so marked an effect. In such urines a slight darkening of color only ensued, much to the same degree as occurred with fresh liquor potassæ. It was never found that liquor potassæ containing lead produced a dark brown coloration with non-albuminous urines, provided, of course, that they were sugar free. The usual slight deepening of the tint took place, but not anything conspicuously greater than with fresh and pure liquor potassæ.

2. *The Fermentation Test.*—When saccharine urine is mixed with yeast and kept in a warm place, it speedily ferments with the production of alcohol and evolution of carbonic acid; and as no other substance is capable of undergoing this transformation, the occurrence of fermentation with yeast is certain proof of the presence of sugar.

Applied to ordinary diabetic urine, fermentation affords very clear indications. The most convenient and elegant way of applying it is the following: A few crumbs of German yeast are put into the bottom of a test-tube; this is then filled up to the brim with the suspected urine, covered with an evaporating dish or saucer, and then inverted. The dish and inverted tube are now set aside in a warm place—say on the mantel-piece. The urine soon begins to ferment, gas collects in the top of the inverted tube and expels a portion of the urine; and if sugar be abundant, the gas accumulates in such quantities that all the urine is driven out before it. There is a precaution, however, to be observed. Some specimens of yeast spontaneously evolve bubbles of gas: it is therefore desirable, where the indication is doubtful, to perform a parallel experiment with the same yeast mixed with simple water, so that the amount of gas spontaneously yielded by it may be ascertained. German yeast is exceedingly convenient for fermentation experiments, and it has now come into such common use that a pennyworth may be purchased in almost any baker's shop.

There are two drawbacks to the clinical application of this test—(*a*) it takes some hours for its accomplishment, and (*b*) it does not suffice for the discovery of minute quantities. Urine is capable of absorbing somewhere about its own bulk of carbonic acid, so that, unless the amount evolved be greater than this, there will be no accumulation of gas in the top of the tube, and consequently no visible sign of fermentation. According to my experience, urine containing 0.5 per cent., or two grains and a half to the ounce and under, yield no sign to the fermentation test. Fermentation is therefore a considerably less sensitive method of sugar-testing than Moore's plan of boiling with liquor potassæ.

There is, however, another manner of applying fermentation to the detection of sugar, which is much simpler and even more delicate than the foregoing—namely, by comparison of the specific gravity of the suspected urine *before* and *after* fermentation. This proceeding will be examined more in detail under the head of quantitative testing; but I may here observe that considerably less sugar than one per cent. may be detected by the lowering of the density after fermentation.

3. *Reduction Tests.*—The action of grape-sugar on a number of metallic salts in alkaline solution is attended by a reduction of the oxides which they contain to a lower degree of oxidation, or to the metallic state. A similar reducing action has the effect of changing the color of several organic solutions. Accordingly some of these substances are resorted to as valuable sugar tests, both qualitative and quantitative.

The metallic salts best adapted for this purpose are those of copper, bismuth, silver, chromium, mercury, and tin; but as the oxide of copper is the most universally known, and with proper precautions the most striking and sensitive, I shall here confine my remarks solely to it.

The ordinary mode of using the copper, or, as it is called, *Trommer's test*, is to add a drop or two of a solution of sulphate of copper to the suspected urine in a test-tube. Liquor potassæ is then added in excess, and the mixture boiled. If the proper proportions have been observed, a red deposit of suboxide of copper falls when sugar is present. Applied in this rough way the operation of the test is very unsatisfactory. If the copper be in excess, a quantity of the protoxide remains undissolved and causes confusion. The liquor potassæ likewise obscures the result by producing an intense dark-brown color if sugar be abundant, and the boiling continued beyond a few seconds. In consequence of these and other objections, Trommer's test is regarded with very little favor by many practitioners, who rely in preference on the easy and ready, though less delicate, method of boiling with liquor potassæ. But all the uncertainty attending the employment of the copper test arises from a faulty application, and not from inherent imperfection. When skilfully used, it possesses a delicacy and certainty that render all other reagents superfluous.

The first necessity is to abandon the rough method above sketched, and to prepare beforehand a test solution which shall combine the copper and the alkali in due proportion. This is accomplished by dissolving sulphate of copper in strong liquor sodæ with the aid of tartrate of potash. The exact formula for this solution (Fehling's standard copper solution) will be given hereafter.

Having prepared the test fluid, it is employed in the following manner: Fill a test-tube to the depth of three-quarters of an inch or so with the copper solution; heat until it begins to boil, and then add *a drop or two* of the suspected urine. If it be ordinary diabetic urine, the mixture, after an interval of a few seconds, will turn *suddenly* of an intense opaque-yellow color, and in a short time an abundant yellow or red sediment falls to the bottom. If, however, the quantity of sugar present be small, the suspected urine is added more freely, *but not beyond a volume equal to that of the test employed.* In this latter case it is necessary to raise the mixture once more to the boiling-point. It is then allowed to cool slowly. If no suboxide has been thrown down when it has become cold, then the urine may with certainty be pronounced sugar-free.

The points of importance in this proceeding are: (*a*) to boil the test first, and not the urine; and (*b*) to use an excess of the test.

The first point is of importance, because the test-solution is apt to deteriorate by keeping, unless preserved hermetically sealed from the air. When deteriorated by exposure to the atmosphere, a deposit of suboxide takes place from it on simple boiling. Boiling the test, therefore, is a trial of its perfection.

If it remain clear for a minute or two after ebullition, the solution is in order, and the testing may be proceeded with; but if the solution become somewhat opaque, and a red deposit presently fall from it, this deposit must be first filtered from the clear fluid, which is thereby again rendered fit for use; or —which is, indeed, the better plan—a fresh supply of the test is prepared. The deterioration here spoken of arises from the conversion of a portion of the tartaric acid into racemic acid, which, equally with sugar, has a reducing power on the oxide of copper, and, when present, of course corrupts the analysis.

The necessity for using an excess of the test applies equally to an ordinary diabetic urine, as well as to one which contains only a small proportion of sugar, and has a composition approaching the natural standard; but as the reason for employing an excess is not the same in the two instances, and as there are important differences in the operation of the test in the two classes of urine, I shall call attention to them separately.

(*a*) *Method of Testing Ordinary Diabetic Urine.*—Practically, the urine of a diabetic patient, where the disease is in full career, may be regarded as a solution of grape-sugar in simple water. The natural constituents are in such small proportion, owing to the increased flow, that they do not sensibly interfere with the operation of the test.

If, after the test has been heated to ebullition, *one* drop of diabetic urine be added, the reaction occurs almost instantaneously, and the suboxide falls of a brick-red color at once; but if *several* drops of the same urine be added, the precipitate is a rich yellow. This difference in color is merely a question of excess or deficiency of the test. When the copper exceeds the sugar, and the solution still retains its blue color, the suboxide falls red; but if the sugar exceed the copper, and the blue color have disappeared, the suboxide falls yellow.

The common mode of proceeding—that is, boiling the urine first, and then adding the reagent—is very objectionable, inasmuch as it may betray the operator into a too sparing use of the test, and thereby entail a failure of the reaction. If the sugar preponderate greatly over the copper, *no precipitation whatever ensues*, because the excess of sugar dissolves the suboxide, and forms with it a transparent yellow solution. This statement may be readily verified by boiling some diabetic urine in a test-tube, and then dropping in the test-solution. The first few drops occasion a dense, muddy, yellow opacity in the topmost layer; but when the tube is shaken the precipitate is redissolved. On adding more of the test, however, the opacity becomes permanent, and an abundant deposit presently subsides.

(*b*) *Method of Testing where the Quantity of Sugar is Small and the Natural Constituents Approximate their usual Proportions.*—The discovery of sugar in such a urine is much more difficult than in the former case. The ordinary ingredients of the urine—urea, uric acid, pigmentary and other extractives, the alkaline and earthy salts—seriously affect the delicacy of the test. If grape-sugar be dissolved in simple water, such is the sensitiveness of the reaction that one grain in ten pints yields a perceptible deposit; but when dissolved in urine, a considerably larger quantity may be present and the test fail to reveal its existence. Nevertheless, enough of delicacy remains to satisfy all the requirements of clinical research. A still greater delicacy can be imparted to the test by the method suggested by Seegen ("Brit. Med. Journ.," 1872, i. 469). The urine is filtered repeatedly through animal charcoal until it is completely colorless—a little distilled water is then passed through the filter, and to this water the test is applied in the usual way. An exceedingly minute trace of sugar (0.01 per cent.) can be detected by this procedure.

Urine of the kind here considered—with a minute proportion of sugar, and the ordinary ingredients almost natural—is met with in the early stage of diabetes, before the disease has acquired its full development; also in convalescence from the less severe forms; and not unfrequently towards the fatal close of the complaint. Even in well-marked diabetes there are conditions under which the urine temporarily returns nearly to its natural state. These are: abstinence from saccharine and amylaceous food, and, *à fortiori*, abstinence from all food; accordingly, the morning urine, after the prolonged fast of the night, may, in the less severe cases, be found almost sugar-free. A like effect follows the advent of an intercurrent inflammation, as of the lungs or lining membrane of the bowels.

In testing for sugar in urines of this description certain precautions are rigidly demanded, otherwise considerable quantities of sugar may be wholly overlooked. The most important of these is to use a great excess of the test. When the copper solution is added drop by drop to healthy urine, at a boiling heat, the blue color is immediately discharged, although not a particle of sugar be present, and the urine assumes a deep amber tint. The degree to which urines exercise this decolorizing property varies with their strength—that is, with their concentration. A dense urine (sugar-free) will discharge the color from nearly its own bulk of Fehling's standard solution; but even the most dilute natural urines—those that are almost colorless—have a very considerable power this way. Whatever be the nature of the transformation here involved, it is certain that when the color of the test has been thus discharged, the copper it contains is no longer capable of being precipitated by any sugar that may be present in the urine; and the suboxide is not thrown down until such an amount of the solution has been added that the mixture retains a distinctly green tint after being raised to the boiling-point. To secure an excess of the test, the most certain method is to heat the solution first, as already recommended, and to add the suspected urine afterwards. Another advantage is secured by this proceeding. When the suspected urine contains a considerable quantity of earthy phosphates, the precipitation of these by the alkali of the test is apt to cause embarrassment. The phosphates fall in light, dirty-white flocculi, which might be mistaken by the unwary for a deposit of suboxide. When the test and urine are mixed together before applying heat, or the test is added to the boiling urine, the earthy phosphates fall in such fine flakes that the transparency of the mixture is impaired; but if the urine be added to the boiling test, the mixture retains its translucency from the phosphates being thrown down in denser masses; and by holding the tube between the eye and the light, the flakes are seen floating in a clear, bluish-green medium.

In the class of saccharine urines now under consideration, the suboxide is always precipitated yellow, never red. The operation of the test is exceedingly distinctive, and takes place as follows: The copper solution having been heated to ebullition, and something less than an equal bulk of the suspected urine having been added, the mixture is again raised to the boiling-point. It then changes to an intense opaque yellowish-green, and slowly a bright-yellow deposit subsides. If the urine contains less than half a grain per cent. of sugar, the precipitation does not take place immediately, but occurs as the liquid cools—in five, ten, or twenty minutes, and the manner of the change is peculiar. First, the mixture loses its transparency, and passes from a clear olive-green to a light greenish opacity, looking just as if some drops of milk had fallen into the tube. This green milky appearance is quite characteristic of sugar. By this proceeding one-tenth of a grain per fluid-ounce, or less than one-fortieth of a grain per cent., can with certainty be detected, and any quantity below this has no pathological signification, and is a matter of only physiological interest.

Some of the natural urinary ingredients, and especially uric acid, have been stated to possess the power of reducing the oxide of copper to a state of suboxide, and of becoming thereby the source of a notable fallacy in using this test for the detection of sugar. In practice, however, no fear need be entertained on this score; I have over and over again treated urines containing an excess of uric acid, and even urines thick with the amorphous urate deposit, with the test-solution at a boiling heat, but have never obtained the least resemblance to the sugar reaction.[1] It is, however, to be borne in mind, that if urine be boiled with the test *for a considerable time*, a reddish deposit falls, and the mixture assumes a muddy, dirty fawn appearance, although no sugar be present. This reddish deposit appears to consist of the earthy phosphates tinged red by some of the suboxide, reduced, perhaps, through the instrumentality of uric acid. But this reddish deposit is *only produced after prolonged boiling*, and prolonged boiling is of all things the most to be avoided, because the most utterly useless, in performing the test. If simply raising the fluid to the boiling-point, and then allowing it to cool in a warm place, as in a jug of hot water or on the hob, fail to yield an indication of sugar, no amount of boiling will develop a trustworthy reaction.

To recapitulate, the best method of detecting sugar in urine is as follows: Pour some of the prepared test-liquid into a narrow test-tube to the depth of three-quarters of an inch; heat until it begins to boil; then add two or three drops of the suspected urine. If the sugar be abundant, a thick yellowish opacity and deposit of yellow suboxide are produced (and this changes to a brick-red at once if the blue color of the test remain dominant). If no such reaction ensue, go on adding the urine until a bulk nearly equal to the test employed has been poured in; heat again to ebullition; and, no change occurring, set aside without further boiling. If no milkiness is produced as the mixture cools, the urine may be confidently pronounced free from sugar, for no quantity above a fortieth of a grain per cent. can escape such a search, and any quantity below that is devoid of clinical significance.

Fehling's test may be rendered portable by compressing the solid ingredients into pellets as suggested by Dr. Pavy,[2] or by enclosing the fluid in glass capsules (Dr. Ralfe[3]). In these ways the stability of the test is also permanently insured.

The Indigo-carmine Test.—If a solution of indigo-carmine (sulph.-indigotate of soda) be rendered alkaline by carbonate of soda and boiled with a small quantity of grape sugar, the indigo-blue becomes reduced to indigo-white, which causes the blue solution to assume a yellow color. This test, originally introduced by Mulder, has been recently recommended by Dr. Oliver,[4] who makes use of it in the convenient form of test-papers. Two test-papers, one saturated with indigo-carmine, and the other with carbonate of soda, are dissolved in a small quantity of water, the result being a clear, blue solution. To this, one drop of the suspected urine is added, and the mixture boiled, when, if sugar be present, the blue color will successively give place to reddish-violet, different shades of red, and finally to a pale yellow tint. On standing, the solution reabsorbs oxygen from the air, and gradually returns to its original hue. In Dr. Oliver's hands, this test has proved not only more convenient, but more delicate than Fehling's.

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[1] Hagen and Müller have found that urine free from sugar may show from 0.087 to 0.37 per cent. of reducing agents. *See* Lancet, 1879, i. p. 606; also Pflüger's Archiv, Bd. xvi. S. 567.

[2] Lancet, 1880, i. p. 172. These "pellets" may be obtained from W. T. Cooper, chemist, 26 Oxford Street, London.

[3] Lancet, 1880, ii. p. 192.

[4] Bedside Urine Testing, 1883.

The Picric Acid Test.—When a solution of picric acid is boiled with grape-sugar, in the presence of liquor potassæ, the yellow picric acid is reduced to picramic acid, which has a deep red color. Dr. G. Johnson[1] has introduced this reaction as a test for diabetic sugar in urine; but the method of applying the test will be more fully discussed, when considering its application to quantitative analysis.

ESTIMATION OF THE QUANTITY OF SUGAR IN URINE. (QUANTITATIVE TESTING.)—In early times medical men judged of the quantity of sugar in diabetic urine by the amount of syrup yielded on evaporation. This was a very rude as well as troublesome proceeding. A much readier and not less precise method was to calculate the sugar from the specific gravity. Dr. Henry drew up a table, which Prout afterward extended and improved, showing at a glance how much solid matter per pint was contained in urines at different densities. When the urine voided amounts to several quarts a day, and the natural urinary ingredients have sunk to a very low proportion, the secretion resembles a solution of grape-sugar in pure water. In this condition the density is a moderately accurate measure of the quantity of sugar; but it is still far from absolute correctness, as may be judged from the following table, drawn up from a number of my analyses:

TABLE *showing the uncertain relation of the specific gravity to the proportion of sugar where the daily flow of urine ranged between nine and thirteen pints.*

Specific gravity.	Sugar per imperial pint.
1045	875 grains.
1048	972 "
1042	683 "
1041	920 "
1041	931 "
1040	911 "
1039	683 "
1035	875 "
1034	645 "
1033	635 "

But when the flow of urine is no more than two or three pints a day, the natural ingredients come to hold something like their normal proportions, and contribute very sensibly to raise he density. Accordingly with the diminished flow there is a very greatly lessened proportion between the specific gravity and the percentage of sugar. The annexed table shows this relation in the urines of the same patients when the daily excretion had been reduced by dietetic means to *between two and three pints.*

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[1] Albumen and Sugar Testing. London, 1884.

TABLE *showing the lessened and still more uncertain relation of the specific gravity to the quantity of sugar where the daily flow ranged between two and three pints.*

Specific gravity.	Sugar per imperial pint.
1044	625 grains.
1042	553 "
1041	591 "
1041	498 "
1039	568 "
1039	608 "
1039	600 "
1039	446 "
1036	377 "
1035	471 "
1034	486 "
1034	312 "

On comparing these two tables, it is seen that the density holds a much less constant relation to the proportion of sugar when the daily flow is scanty than when it is abundant. It is also seen that in the former case a given degree of density indicates a much lower proportion of sugar than in the latter. The mean density in the first table is 1039.3, and in the second nearly the same—1038.6; but the proportion of sugar is much greater in the first, where it averages 813 grains per pint, than in the second, where it is only 511 grains.

Of the more accurate processes there are two peculiarly eligible for practical use—the one on account of its speedy performance, and the other on account of its easy application.

1. *Volumetrical Processes.*—These depend in principle on the fact that there is a fixed relation between the amount of sugar present and the amount of metallic salt or picric acid reduced by its action. Thus, Fehling found that one molecule of grape-sugar, or 180 parts, decomposed exactly five molecules, or 1246.8 parts, of sulphate of copper. Accordingly he prepared a solution of copper of standard strength, and applied it to fluids containing grape-sugar; and the quantity of these required to decompose a fixed volume of the standard solution furnished an exact measure of the sugar they contained.

The solutions which have been used for the estimation of sugar in the urine are those of sulphate of copper, either in Fehling's method, or in the modification of it suggested by Dr. Pavy, and of picric acid, as applied by Dr. G. Johnson.[1]

a. Fehling's Method.—Fehling's standard solution is prepared according to the following prescription:

Crystals of sulphate of copper	34.64 grammes, or	90½ grains.
Neutral tartrate of potash	173 "	364 "
Solution of caustic soda of sp. gr. 1.12	480 c.cm., or	4 fluidounces.

Add water to make up 1000 cubic centimetres or 6 fluidounces.

Every 10 cb. centim. corresponds to 0.05 gramme of grape-sugar, and 200 grains to 1 grain of sugar. The apparatus required for the performance of the analysis is described and figured at pp. 36 and 37.

Mode of Performing the Analysis.—Measure off 200 grains of the standard solution in the 200-grain tube, pour this into the flask, and add about twice its volume of water; then place over a spirit-lamp to boil. While the copper solution is being heated, the urine to be analyzed should be diluted with water to a known degree. In the case of ordinary diabetic urines, the best dilution is one in ten. This is obtained by carefully filling the 6 oz. measure with water to the depth of 4½ ounces, and then adding urine so as to make up exactly 5 ounces. The mixture will then contain exactly one-tenth of urine. (When the quantity of sugar in the urine is very small, a dilution of one in five, or even the undiluted urine, may be employed.)

The next step is to fill the burette (which is graduated to grains) with the diluted urine to 0. Then proceed to add it, in successive small portions, to the boiling copper solution, until the blue color has entirely disappeared. After each fresh addition from the burette the mixture should be raised to the boiling-point, and then allowed to stand a few seconds, so that the precipitated copper may subside, and the observer may see, by holding the flask between the eye and the light, whether the mixture still retains any blue color. As soon as the blue color has disappeared the analysis is complete, and the quantity of diluted urine employed may be read off. The percentage of sugar in the urine can now be readily calculated. Suppose 125 grains had been added from the burette; this represents one-tenth, or 12.5 grains, of undiluted urine, and contains exactly one grain of sugar; by dividing 12.5 into 100, the percentage of sugar in grains is obtained; or $\frac{100}{12.5}=8$; the urine contains 8 per cent. of sugar.

If the metrical system is preferred, the process would be carried out in the following manner: Into a burette graduated in cubic centimetres, 10 cubic centimetres of urine are placed and diluted to 100 cubic centimetres, with water. Place in the flask 10 cubic centimetres of the Fehling's solution (representing 0.05 gramme of sugar), and dilute with about twice its volume of water; then boil as before. The amount of diluted urine required to reduce the copper in the Fehling's solution is then determined, and the calculation made as follows: Suppose that 12 cubic centimetres of the diluted urine were required; this would represent 1.2 cubic centimetre of urine, which would contain 0.05 gramme of sugar. The number of grammes contained in 100 cubic centimetres of urine would be obtained by the proportion:

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[1] Knapp's method, by which the reduction of cyanide of mercury to the metallic state is applied for the estimation of sugar, is not recommended for urine testing.

$$\frac{1.2}{0.05} = \frac{100}{x} \therefore x = \frac{100 \times 0.05}{1.2} = 4.16.$$

To determine more exactly the point at which the whole of the copper has been reduced, when the end of the reaction is believed to have been reached, a portion of the fluid in the flask may be filtered. The filtrate should be quite free from blue color, and if acidified with acetic acid and a solution of potassium ferrocyanide added, no brown color should be produced. If, by these means, copper should be found in the filtrate, sufficient urine has not been added, and the process must be repeated from the beginning. On the other hand, if a few drops of Fehling's solution added to the filtrate should give a deposit of copper oxide on boiling, too much urine has been added, and the process must again be repeated. For clinical purposes, however, the disappearance of the blue color can be determined by the eye with quite sufficient exactitude.

b. Dr. Pavy's Method.—Dr. Pavy has introduced a modification of Fehling's method, by which the reduced oxide of copper is retained in solution by ammonia. The action of the sugar is then shown, not by precipitation of the copper oxide, but simply by the discharge of the blue color of the solution. The solution recommended by Dr. Pavy is composed as follows:

Cupric sulphate	4.158	grammes.
Potassic sodic tartrate	20.4	"
Potash (caustic)	20.4	"
Strong ammonia (sp. gr. 0.880)	300	c.cm.

Water to 1 litre.

Of this solution 10 cubic centimetres are decolorized by 0.005 gramme of sugar. The test may also be kept either in the form of pellets or enclosed in glass tubes each containing 10 cubic centimetres. To make the solution the tartrate of potash and caustic potash are dissolved together in one portion of the water and the sulphate of copper in another portion. The solution of sulphate of copper is then poured into that of the potash salts, and to this the ammonia is added, the whole being diluted with water to the required amount. The pellets are of two kinds—one containing the tartrate of potash and copper sulphate together with ammonium chloride, the other containing the caustic potash. The two pellets are dissolved separately and the solutions mixed, when the whole will represent 10 cubic centimetres of the original solution. To apply the test a burette is filled with the urine to be examined, diluted to—preferably—1 part in 20. There must now be placed in a small flask, 10 cubic centimetres of the copper solution diluted with 20 cubic centimetres of water, and the whole raised to the boiling-point. The diluted urine is then run in from the burette drop by drop, until the whole of the blue color has disappeared from the solution. The amount of diluted urine thus required, divided by twenty, will give the number of cubic centimetres of urine containing 0.005 gramme of sugar. During the process air must be excluded from the flask, for the colorless solution of cuprous oxide in ammonia soon absorbs oxygen, forming again the blue solution of cupric oxide. For details of the process and for a convenient table, giving the amount of sugar per 1000, corresponding to the number of cubic centimetres of urine used, the reader is referred to Dr. Pavy's paper in the "Lancet," I. p. 376, 1884.

The process has the advantage of determining the end-point of the reaction more easily than can be done by Fehling's method, and the ammoniated solution is also more stable than Fehling's solution.

c. Johnson's Method.—As previously mentioned, this test depends on the power of grape-sugar to reduce a yellow solution of picric acid, in the presence of caustic potash, to a red solution of picramic acid,—the depth of the red color depending on the amount of sugar present. In applying the test a standard solution of picramic acid is required, with the color of which, the result of boiling the urine with picric acid and potash may be compared. A standard solution representing the color produced by the presence of ¼ grain of sugar to the ounce of urine, is found convenient; but since the solution of picramic acid is liable to change rapidly on exposure to light, it is better to keep as a standard, a solution of acetate of iron, which, if made according to the following formula, will equal in color the standard picramic acid solution.

Liq. ferri perchlor. fort.	(sp. gr. 1.338)	1	drachm.
Liq. ammon. acetat.	(sp. gr. 1.017)	4	"
Glacial acetic acid	(sp. gr. 1.065)	4	"
Liq. ammoniæ	(sp. gr. 0.959)	1	"

Distilled water to 4 ounces.

In this method also, it is desirable to dilute the urine, before examining—say to five or ten times its volume. A drachm of the diluted urine is then taken and boiled for sixty seconds with 30 minims of liq. potassæ and 40 minims of concentrated solution of picric acid, sufficient water being added to make four drachms of the solution. At the end of the boiling, if the solution is found to be less than four drachms, it must be raised to that amount by the addition of more water. This part of the process is conveniently carried out in a long test-tube marked at the height of four drachms. The deep red solution must now be diluted until its color exactly equals that of the standard solution. This is done in a stoppered tube, twelve inches long and three-quarters of an inch in diameter, graduated into 10 and 100 equal divisions. Attached to this tube is another smaller tube, containing the standard solution. 'A quantity of the boiled urine and test, is poured in a large tube, until the tenth division is reached and distilled water is added carefully until the colors of the liquid in the graduated tube and that in the smaller tube are exactly alike. The number of degrees of dilution required to produce this is then read off. During the boiling the urine was diluted four times, and hence, if it then just equalled in color the test solution it would have contained one grain of sugar to the fluid-ounce. If further dilution were required to bring it to this standard, say from ten to thirty-five divisions, 3.5 grains to the ounce would be indicated.

Previous to the admixture with the test, however, the urine was diluted—say ten times. Hence, the result above obtained must be multiplied by ten, to obtain the number of grains of sugar per ounce of the original urine.

A full account of the method will be found in Dr. Johnson's treatise on "Albumen and Sugar Testing," London, 1884. The apparatus required are made by E. Cette, 36 Brooke Street, Holborn, E.C.

2. *Differential Density Method.*—This method of estimating sugar combines, as I believe, more perfectly than any other, the twin advantages of ease and accuracy. It is founded on the diminution of density suffered by saccharine urine when fermented with yeast. The specific gravity of an ordinary diabetic urine ranges from 1035 to 1050. When it has undergone fermentation, and all the sugar is converted into alcohol and carbonic acid, the specific gravity is found to have sunk to 1009, to 1002, or even below 1000. This falling off in the density arises from two distinct yet necessarily associated causes—namely, first, the destruction of the sugar, which was the cause of the high density of the original urine; and, second, the presence of the generated alcohol in the fermented product. Now the loss of density from these causes must evidently stand

proportional to the quantity of sugar originally present in the urine, and must consequently furnish a measure of its quantity.

The experimental data on which this method is founded are fully detailed in a paper published by the author in the "Memoirs of the Manchester Literary and Philosophical Society" for 1860; also in a paper in the "Edinburgh Monthly Journal" for October, 1861. The mode of experimenting was —first to ascertain by Fehling's method how much sugar was contained in a certain diabetic urine. The urine was then fermented by means of German yeast—its specific gravity having been previously ascertained. In twenty-four hours, after the fermentation had ceased, and the scum had subsided, the density was taken again, and by subtracting this from the density before fermentation, the "density lost" was ascertained. And it was found that for every grain of sugar contained in an ounce of urine, one degree of specific gravity had been lost. Experiments were multiplied on diabetic urine; corresponding experiments made with solutions of sugar of known strength in healthy non-saccharine urine and in pure water, and the issue of all was to establish the conclusion that *the number of degrees of "density lost" indicated as many grains of sugar per fluid-ounce.*

In the practical application of the method, the ordinary urinometer may be used for taking the densities; but it is well to choose one with a long scale, as some of those in use have very short ones, and it becomes impossible to read the density accurately. Still further precision may be attained by dividing the usual scale into two parts on separate instruments. I have had constructed for my own use two perfectly corresponding urinometers, on one of which the scale ranges from 995 to 1025, and on the other from 1025 to 1055, each instrument covering 30 degrees of density. The scales are thus rendered so long, and the intervals between the lines so great, that in a clear urine the specific gravity can be easily read to a quarter of a degree; and even in fermented urine, which does not regain its original transparency, but continues, at least for many days, more or less cloudy, it can be read with certainty to half a degree.

Another important point is to obviate errors from variations of temperature. If the density before and after fermentation be taken at widely different temperatures, an error of serious amount may creep into the analysis. The best mode of avoiding this is to put up a few ounces of the unfermented urine in a "companion phial," and to place this side by side with that set apart for fermentation, so that, at whatever temperature the fermented product may be when its density is observed, its unchanged *alter ego* stands near it for comparison at exactly the same temperature.

The most convenient way of proceeding is the following: About four ounces of the saccharine urine are put into a 12-ounce bottle, and a lump of German yeast about the size of a cobnut or small walnut is added to it. A great excess of yeast is used to hasten fermentation, but a little more or a little less does not sensibly affect the result. The bottle is then covered with a nicked cork (which permits the escape of the carbonic acid), and set aside on the mantel-piece or other warm place to ferment. Beside it is placed a tightly corked 4-ounce phial filled with the same urine without any yeast. In about twenty-four hours the fermentation will have ceased, and the scum cleared off or subsided. The fermented urine is then decanted into a urine-glass, and its specific gravity taken; at the same time, the density of the unfermented urine in the companion phial is observed, and the "density lost" ascertained. Fermentation is generally complete in about eighteen hours, if the locality be sufficiently warm; and it is desirable to remove the two phials into a cool place two or three hours before the densities are taken, in order that they may attain the temperature of the surrounding atmosphere.

The two following examples may serve as illustrations of the method:

	I.	II.
Density before fermentation	1053	1038
Density after fermentation	1004	1013
Degrees of density lost	49	25
Grains of sugar per fluid-ounce	49	25

If it be desired to bring out the result as so much per cent., this is accomplished by multiplying the number indicating the "density lost" by the coefficient 0.23. Thus in the first of the above examples $49 \times 0.23 = 11.27$, and in the second $25 \times 0.23 = 5.69$, which are the amounts of sugar respectively per 100 parts.

The time actually consumed in determining the quantity of sugar in urine by this method does not exceed four or five minutes, but the result must be waited for until the succeeding day; this is its chief advantage. Its application is so easy, that a medical friend in attendance on a diabetic patient was able to teach the patient's wife to make the analysis; every morning when he came, she could give exact information as to the quantity of sugar excreted on the previous day.

Dr. Hensley[1] has investigated the limits of error which the use of this method involves. He finds that the rule arrived at experimentally—namely, that one degree of density lost in fermentation corresponds to one grain of sugar per ounce of urine

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[1] Note on Dr. Roberts's method of estimating diabetic sugar, by Philip J. Hensley, in vol. iii. of the St. Bartholomew's Hospital Reports.

—agrees very closely with the theoretical result obtained by calculation. Dr. Hensley concludes that if the above rule be applied to the fluid-ounce measure of the British Pharmacopœia, and sugar be taken as dry crystallizable glucose ($C_6H_7O_7$), the result obtained is slightly too large, but the excess above the true number is less than its sixty-fourth part, that is to say, less than 1.6 per cent.

Optical Saccharimetry.—The property of glucose of rotating the plane of polarization to the right has been taken advantage of to estimate the quantity of sugar in diabetic urine. The best instruments for the purpose are those of Mitscherlich and Soleil. This method is not so universally applicable as the preceding; and the price of the instruments, together with the delicacy required in their manipulation, puts them almost out of reach of ordinary practitioners.

CLINICAL SIGNIFICANCE OF SUGAR IN THE URINE.—The presence of a large quantity of sugar in the urine is the characteristic feature of diabetes mellitus: but small quantities may be present in a variety of other circumstances—as after eating excessively of amylaceous or saccharine articles of food, from injury or disease of certain parts of the nervous system, from impediments to respiration, etc. This subject, however, can be more conveniently treated in a future page (*see* Physiological Considerations relating to Diabetes).

PART II.

URINARY DISEASES—DISEASES OF WHICH THE CHIEF CHARACTERISTIC IS AN ALTERATION OF THE URINE.

CHAPTER I.

DIABETES INSIPIDUS.

Cases characterized by increased thirst and excessive discharge of a watery urine of low specific gravity, free from sugar and albumen, are grouped together under the general designation of diabetes insipidus.

The want of uniformity in the course and symptoms of these cases, and in the anatomical changes found after death, indicate that several wholly distinct pathological states are included under this heading.

Attempts have been made to classify the cases according to the characters of the urine. Those in which it was supposed that the urine merely contained an excessive amount of water, without any alteration of the total quantity of solids excreted, or of the mutual proportion of the several solid ingredients to each other, have been named *Polydipsia* (or excessive thirst); those in which it was supposed that the solid matters, and especially urea, were excreted in excessive quantity, have been named *Polyuria;* and those in which it was supposed that the urea and other solids were in diminished quantity, have been named *Anazoturia* (Willis).

This classification is, however, valueless in practice: both from the difficulty of assigning a precise standard of composition to the urine under the various conditions of existence, and the tedious and difficult investigations, extending over several days, which are required to ascertain the mean composition of the urine in any particular case.

The following account of diabetes insipidus has been drawn up from an analysis of one hundred and twenty cases,—either collected from various sources or observed by myself.

ETIOLOGY.—The liability to diabetes insipidus is very considerably greater in males than in females; of one hundred cases, seventy-two were males, and twenty-eight females; the *age* of the patients at the time of invasion ranged from the extremes of infancy to old age; but the greater number occurred between the ages of five years and thirty years. In the following table an analysis is given of the ages of seventy cases at the time of invasion.

Infancy	7 cases.	From 20–30 years	16 cases.
From 5–10 years	15 "	" 30–50 "	15 "
" 10–20 "	13 "	" 50–70 "	4 "

In two, if not three, cases the disease appeared to have existed actually from birth.

In a very large proportion, no *exciting cause* whatsoever could be assigned for the disorder. In the remainder, various circumstances were alleged with greater or less probability to have been the exciting causes. These present considerable similarity to the alleged causes of saccharine diabetes, and stand in the following order of frequency:

Cerebral disease (tubercle, etc.)	11	Previous febrile or inflammatory disease	6
Blows on the head, and falls	8	Hereditary influence	3
Intemperance	5	Muscular effort	2
Exposure to cold, and drinking cold fluids while heated	5	Exposure to hot sun	1
		Mental emotion	1

In several cases serious organic changes were found in the kidneys. These will be more particularly described in connection with the morbid anatomy of the disease.

Two cases recorded by Dr. W. Watts ("Lancet," 1848) are referred by him to syphilitic disease and abuse of mercury.

Hysteria, grief, neuralgia, or the influence of a nervous constitution, are also mentioned as determining causes.

In some of the traumatic cases the symptoms set in with maximum intensity on the very day of the accident; in others there was at first loss of consciousness; and the thirst and diuresis came on with the restoration of the faculties, or a few days after. In one case severe nervous symptoms continued for six months after a fall, and the diuresis first broke out at the end of this period. In four of the traumatic cases the symptoms persisted for between nine days and a month, and then finally disappeared as the cerebral symptoms subsided; in two others the disorder became permanent, and had already existed at the date of the record, six years in one and seven years in the other.[1]

In the cases associated with cerebral disease, tumor of the brain was found in four—and degeneration of the cell-elements in three others. These cases will be noticed more particularly when the morbid anatomy of the disease comes to be treated of. In a case observed by myself (a shopkeeper thirty-five years of age), the disease had come on twenty months previously with sudden, complete, and permanent loss of sight, first in the left eye, and six months later in the right. During these twenty months the patient had been in the habit of voiding two or three gallons of urine daily. He was also subject to curious nervous attacks, which recurred at irregular intervals, and lasted from half an hour to periods of several days. They consisted in a perversion of intellect, incoherence, irrepressible impulse to go away from the house, trembling of the limbs and twitching of the muscles. Sometimes the patient would fall into an epileptiform fit, with loss of consciousness, screaming and convulsions, but without foaming at the mouth, or biting the tongue. When seen by me he was totally blind, but the intellect was perfect, and the general health—except during the paroxysms—was good. He could walk twelve miles with ease; and in the last eight months he had gained weight to the extent of 40 lbs. The history and general character of the symptoms appeared to point to the existence of vesicular parasites within the cranium. The three remaining cases of this group were children supposed to suffer from cerebral tubercle. They all died in convulsions.

Of the five cases attributed to intemperance, the symptoms came on in one of them on the day after a severe bout of drinking, in which the patient had been insensible for two days. Not one of this group is reported as cured; and one died in two months.

Two cases followed exposure to cold; and two followed copious drinking of cold fluids while the skin was hot and perspiring. One of the latter, related by Vigla, began with unquenchable thirst and diuresis on the same day, and terminated fatally a few months after.

Four cases followed variola, ague, fever, and inflammation of the bowels; all ran a very chronic course, and lasted from four to twenty-four years, with good preservation of health; the symptoms commenced immediately after recovery from the initial complaint.

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[1] Dr. Matthews Duncan has sent me the notes of a case (read before the Edin. Obst. Soc., June 10, 1874) of D. Insipidus, in a woman who miscarried at the seventh month. Fourteen years before she had a blow of great severity on the back of the head. Shortly after this she observed that she drank a great deal of water, and had a great flow of urine. This has continued ever since, though she has enjoyed fair health, and is the mother of four healthy children. The quantity of urine ranged from 20 to 30 pints.

In two cases, the symptoms commenced immediately after violent muscular effort. One was a boy of twelve, who strained himself in pushing a cart-wheel sunk in the mud. After a few months, the symptoms were subdued by nitrate of potash; but some months later a relapse occurred, and the patient died suddenly, from taking, as is alleged, too large a dose of the nitrate (P. Frank—cited by Romberg). The second is a remarkable case, related by Jarrold, in Duncan's "Annals" for 1801. A girl of 19, when going down a flight of steps, slipped; with very great exertion she saved herself from falling. Immediately after menorrhagia began, and on the evening of the same day she experienced inordinate thirst and profuse diuresis. She entered the Edinburgh Infirmary, under Professor Gregory, and was speedily cured of the hemorrhage by the compound powder of alum. The urine amounted to the enormous quantity of 50 lbs. in the twenty-four hours, sometimes even to 60 lbs., and one day to 72 lbs.! Under the influence of lime-water and powdered galls, the urine was gradually reduced to between 5 lbs. and 10 lbs. a day. She left the hospital otherwise in good health.

Three cases were attributed to hereditary influence. One of these was a man in good health, who had suffered for the long period of fifty-nine years from polyuria. The disorder began in infancy. His father, two brothers, and a sister had suffered similarly. Another was a healthy soldier of twenty-four, who had been polyuric for four years. His mother, brothers, and two sisters suffered in the same way. The third was a young lady of nineteen, mentioned by Trousseau ("Clinique Médicale," t. ii. 611), whose grandfather was affected with saccharine diabetes, and uncle with Bright's disease. She was well-grown and tolerably healthy, and had borne her complaint for six years. All these cases proved incurable.[1]

COURSE AND SYMPTOMS.—The invasion of the complaint is often quite sudden. Dr. Bennett relates the case of a woman, thirty-four years of age, who went to her work one morning at six o'clock in her usual health; at eight o'clock, two hours after, she was suddenly seized with intense thirst and diuresis, which became persistent from that time.

In several instances it is recorded that an intercurrent febrile or inflammatory disorder temporarily suspended the symptoms.

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[1] Orsi (Virch. and Hirsch. Jahresb., ii. p. 244, 1881) reports a most remarkable family history. In a family of nine, consisting of parents, four sons, two daughters, and a maternal uncle, no less than six suffered from Polyuria. Weil (Virch. Archiv, Bd. 95) gives a still more remarkable history of a family, in which no less than twenty-two members were the subjects of Diabetes Insipidus.

In one case, an attack of acute articular rheumatism (treated with nitrate of potash) suspended the disease permanently, after it had existed in intensity for eighteen years. In another instance (a girl of nineteen, polyuric from infancy), an attack of pleurisy was treated by a blister, which suppurated for thirty-five days; at the end of this time, both the pleurisy and the polyuria disappeared permanently. In a third case (recorded by Külz) an attack of varioloid suspended the symptoms temporarily during the course of the fever.

The quantity of urine voided by persons afflicted with insipid diabetes, is usually considerably greater than in saccharine diabetes; 15, 30, and even 40 pints are frequently mentioned as the daily amount of urine.[1] Its specific gravity varies from a little above that of pure water to 1003 and 1007. It is limpid and colorless, and contains but a feeble proportion of solid matters. The total quantity of urea excreted in twenty-four hours is usually greatly increased. In many cases also the amount of phosphates is much in excess of the normal,[2] but the proportion to each other of the remaining normal ingredients of the urine has not been found sensibly altered. The only abnormal substance that has hitherto been detected is inosite. Inosite in small quantities has been found several times; and it has been suggested that the presence of this substance in the urine was characteristic of the disease. This is, however, not the case. Inosite has been found repeatedly in D. Mellitus and in Albuminuria. Probably, as Strauss conceives, the appearance of inosite is merely a coincidence of the excessive transudation of watery fluid through the tissues of the body; for he found inosite in the urine of three healthy persons who, for the purpose of experiment, had drunk in the course of a day a large quantity (10 litres) of water.[3]

The thirst is generally intense; often inextinguishable; in several cases the patients are stated to have drunk their own urine. When the quantity of drink and the quantity of urine were compared, sometimes the one and sometimes the other showed in excess. Careful determinations on this point by Falck, Neuschler, and others, indicate that if fluids be allowed *ad libitum* the urine voided is about the same quantity as the drink; but if the imbibition of fluids be compulsorily diminished, the urine is not diminished in the same proportion, and dehydration of the tissues results.

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[1] A little girl of ten, under my care at the Manchester Infirmary, passed rather more than a third of her own weight of urine daily for some weeks—and yet continued in fair health. Her weight was 56 lbs.

[2] *See* Dickinson, loc. cit., and Ralfe, Lancet, i., 1881. This phenomenon, however, is hardly sufficient to justify the term Phosphatic Diabetes, as introduced by Teissier.

[3] *See* Gallois's Thesis, De l'Inosurie, Paris, 1864; and Strauss's Thesis, p. 25.

The skin is generally dry and harsh; sometimes it preserves its natural moisture, and in rare examples sweating has been observed. It is noteworthy that boils and carbuncles are rarely mentioned.

Külz records a case in which spontaneous persistent ptyalism (not caused by mercury) coexisted with D. Insipidus in a girl of eighteen. She was under observation for about four months; and voided daily from 12 to 18 ounces of saliva and 200 to 260 ounces of urine. Külz calls attention to the fact that ptyalism is produced in dogs and rabbits (according to the experiments of Eckhard, Nöllner, and himself) by puncture of the floor of the fourth ventricle, showing that the controlling nerve-centre of the salivary glands lies closely adjacent to that of the kidneys, and supplying the physiological key to the coexistence of D. Insipidus and ptyalism. Worm Müller also has reported a case in which the saliva was much increased in quantity.[1]

The state of the general health varies a good deal. In the greater number of the recorded cases fair health was preserved —in several patients the health was perfect, and some of them became fathers and mothers of families, and went about their usual avocations without other detriment than the inconvenience of a constant thirst and incessant calls to void urine. A remarkable example of this kind was communicated by Mr. Maxwell to Dr. Simmons ("Med. Facts and Obs.," vol. ii. 96). A hale farm laborer, aged fifty-one, who habitually performed the severest tasks, thrashing, mowing, etc., like his fellow-workmen, had been polyuric for twenty-four years. The disorder came on after a fit of ague. The patient drank daily, summer and winter, from 32 to 36 pints of water, and voided urine in proportion. Yet he slept well (except that he frequently awoke to drink): he had no pain or ache of any sort; he had an excellent appetite, a moist skin, and perspired freely when he was at work. Dr. Simmons also cites the case of a woman residing in Paris, who had been polyuric from infancy. In due time she married a cobbler, and became the mother of eleven children, of whom, however, only two were living when the case was recorded. Dr. Willis quotes the history of an artisan, aged fifty-five, who entered the Hôtel Dieu, of Paris, for some trifling bruise of the knee, from which he speedily recovered. From the age of five years he had suffered from a constant thirst, accompanied with a commensurate diuresis. From his sixteenth year he had drunk on an average two bucketsful of water daily. This man continued in good health; he was the father of several children, and experienced no inconvenience from his infirmity beyond what was inseparable from the frequent calls to pass water, and the constant necessity for drink. In a boy of ten, under my care, in the Manchester Infirmary, who had been voiding about fifteen pints of urine daily for several months, the general health and nutrition were perfect. Excepting a dry tongue and skin, there was no abnormal condition apart from the excessive thirst and polyuria. The boy was active and intelligent, and he ate and slept well, and looked rosy and plump.

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[1] See Virch. and Hirsch. Jahresber., 1879, ii. p. 246.

This high state of health is however exceptional: more commonly the patients are very decided valetudinarians; and the symptoms from which they suffer bear a resemblance to those of diabetes mellitus, though rarely exhibited in equal severity. These are epigastric and lumbar pains; dry, harsh, hot skin; painful dryness and heat of the mouth and fauces; emaciation. Sometimes the appetite is voracious, more commonly moderate or indifferent. The temper is querulous; the mental faculties enfeebled; the bodily strength diminished; the sexual functions often abolished. The face is subject to erythematous congestion. Enforced abstinence from fluids aggravates most of these symptoms: the body then becomes unbearably hot, the skin suffused, a sense of intolerable sinking, or even of intense pain, is felt in the pit of the stomach, and the intellect becomes confused.

The loss of rest, the tormenting thirst, the mental worry, at length produce, in most instances, an exhaustion of the bodily vigor; œdema of the feet often appears towards the last. The disease is, however, seldom fatal directly by its own virulence. More frequently the patient succumbs to some concomitant disorder—phthisis, pleuro-pneumonia, or organic disease of the brain.

In some cases there was dislike to vegetable aliments, in others to animal food. The cobbler's wife, before alluded to, was very sensitive to alcoholic drinks: a single glass of wine caused uneasy sensations in all her limbs, and a sense of faintness. In other instances the patients drank freely of wine or beer, as their condition allowed. In a man observed by Trousseau, there was a remarkable tolerance of alcoholic stimulants. This man on one occasion drank a litre (a pint and three-quarters) of brandy in two hours; and while in hospital he imbibed daily a similar quantity without the smallest inconvenience. The patient related, that since his illness began he had acquired this singular immunity from the causes of drunkenness. More than once he had laid wagers to drink twenty bottles of wine at a single sitting, and had won his wagers without the least disturbance of the nervous system.

Irritability of the bladder, with excessively frequent micturition, was noted in several instances.

The *duration* of the complaint is exceedingly uncertain.

The traumatic cases generally only lasted a few weeks or months: on the other hand, one of the congenital cases had endured fifty-nine years, another fifty years, at the date of the record.

Out of seventy-seven cases collected, sixteen were reported as complete recoveries; fourteen ended fatally; and the remaining forty-seven were still in progress when reported; though, in some of them, considerable amelioration had taken place. In the sixteen recoveries the duration of the disease was mostly comparatively short,—in nine, it was under a year; in one, four years; in two, eighteen and nineteen years; and the remainder "some" years. In the fourteen fatal cases, the duration was still shorter. In nine of them it was under a year; one died in the short space of seven weeks; two more in two months. The other five survived for periods varying from eighteen months to twenty years.

Of forty-seven cases still in progress when reported, the duration of the disease was mentioned in thirty-five instances: Five had continued for a year or under; five, for between one and two years; twelve, for between two and six years; six, for between six and twelve years; four, for between twelve and twenty-four years; and four, for between twenty-four and fifty-nine years.

MORBID ANATOMY.—The condition of the organs after death from diabetes insipidus, has only been ascertained in a few cases. I have collected fourteen *post-mortem* examinations; and to these I add one performed by myself. In three of these cases the lesions found presented a tolerably close similarity, and consisted of an atrophied and degenerated condition of the renal substance; in a fourth, the glandular tissue of the organs was entirely wanting; in a fifth, multiple abscesses were found in the kidneys; in my own case, and in three others, the kidneys were simply hyperæmic and somewhat enlarged, and a tumor was found in the brain.[1] In two cases, fatty degeneration of the nervous tissue of the walls of the fourth ventricle was found.

As these cases are so few in number, I shall describe them more fully.

CASE 1. (Dr. Eade—Beale's "Archives," 1861, p. 8.)—A man, aged 65, had suffered from jaundice and neuralgia; he succumbed in eighteen months to the continual diuresis, and the urgent and incessant calls to void urine. The quantity of urine varied from three to six pints; specific gravity never exceeded 1008; it was free from sugar, albumen, or other morbid ingredient. The autopsy revealed the following: "The infundibula and pelvis of both kidneys were greatly dilated, and the state of sacculated kidney was evidently in process of establishment. Left kidney of natural size. Right, one-half larger, and of darker color. Both showed depressions along the surface, marking the interlobular portions. Previous to section, the cones could be distinctly felt as much denser than the interpyramidal portions, giving indeed the sensation of so many little tumors or nodules. On section, both were seen to be pale and flaccid, and evidently undergoing a gradual process of absorption." The bladder was somewhat large and thickened; the ureters dilated. The thoracic and the other abdominal organs were not diseased.

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[1] In an appendix to the present chapter reference is made to some cases of polyuria (with records of post-mortem examinations), in which a minute quantity of sugar existed temporarily in the urine.

CASE 2. (Dr. Eade—Beale's "Archives," 1862, p. 128.)—A man, aged 62, had experienced excessive thirst and diuresis for twenty years. Health fair, until two years before death, when it began to fail, and for the last nine months he was unable to work. The quantity of urine often amounted to between fourteen and sixteen pints, and had never contained sugar or albumen. There was little pain beyond a sense of weariness. The bowels were constipated, and the stomach very irritable with frequent vomiting. At length the bladder became unable to expel its contents, and a typhoid state supervened; the stomach rejected everything, and he died exhausted.

Autopsy.—Both kidneys were diminished in size, deeply lobed on the surface, and very dense to the feel in the position of the cones. On section, they were seen to be greatly wasted. The cortical portions very thin, and scarcely to be distinguished from the pyramidal. The cones were nearly absent, or rather were converted into dense fibrous tissue, containing many large cystiform spaces. The mucous membrane of the pelvis was thickened, fibrous-looking, and darkly congested. The pelvic cavities considerably enlarged. Ureters a little dilated. On microscopical examination (by Dr. Beale), many of the tubes were found narrow and much wasted, while others were twice their natural diameters. The walls of the tubes were firm and thick. The capillary vessels everywhere were surrounded by a considerable quantity of fibrous material with numerous nuclei. The Malpighian bodies were, for the most part, smaller than in health. The epithelial cells were also smaller, as well as more numerous than in health, and the tubes appeared to be distended in many places by their accumulation. The suprarenal capsules were greatly diseased, and converted into flaccid cysts. The bladder was enlarged, and its walls thin and pale. The other abdominal organs were healthy, except perhaps the liver, which was intensely congested. In neither case does the brain appear to have been examined.

CASE 3. (Neuffer—cited in Magnant's Thesis.)—A man, aged 28. The disease came on after a drunken bout. There was intense thirst; the urine amounted to thirteen or fourteen pints a day; specific gravity 1001 to 1002; without trace of albumen or sugar. He emaciated rapidly; had pain in the epigastrium; at length frequent vomiting; itching of the skin, which was dry; enfeebled vision. He died in about two months.

Autopsy.—The gastric mucous membrane was pale and swollen; the kidneys were notably diminished in size, pale, anæmic; the epithelium of the tubes fatty; bladder contracted; mucous membrane a little tumefied; other organs healthy.

CASE 4. (Dr. Strange—Beale's "Archives," 1862, p. 276.)—The patient was a farm laborer, aged 18, who presented the appearance of a moderately stout lad of 15. He was admitted into the Worcester Infirmary on October 19, 1861. The skin and tongue were natural, and the face ruddy; appetite normal; thirst constantly excessive; bowels generally relaxed. The urine amounted to about twelve pints in the twenty-four hours; its specific gravity was 1007; it contained neither sugar nor albumen. All the history obtainable was, that the patient had been a delicate and backward boy; that he had had this diuresis for a number of years, and that the medical attendant had always affirmed that the urine did not contain sugar.

Dr. Strange, being desirous to ascertain whether the diuresis was kept up by the excessive imbibition of fluids (in accordance with the theory of Prof. Bennett and others), restricted the patient to a more moderate allowance of fluids. A warm bath was administered twice a week. Four days after admission (October 23), the urine measured nine pints; its specific gravity was 1006. On the 26th the bowels were much relaxed; urine five pints. On the 28th, a phosphoric acid mixture which he had been previously taking was omitted, and Mist. cret. co. given instead. On this day the patient complained for the first time of headache, with weakness and loss of appetite; there were also some febrile symptoms. On the 29th, the bowels being still relaxed, five minims of tinct. opii, and half a drachm of tinct. catechu were added to the mixture. On the 30th he became drowsy, with pain at the back of the head; the diarrhœa continued, with vomiting. Effervescing draughts, with nitric ether, were now administered in lieu of the previous medicines; half an ounce of brandy was given three times a day, and cold applied to the head. On November 2d, the drowsiness and sickness had abated; the bowels were confined; the urine three and a half pints, specific gravity 1004. The brandy was omitted, and half an ounce of castor oil administered. As it now appeared that restricting the patient in his drink had resulted in mischief, he was allowed to take as much water or barley-water as he pleased. On November 4th, in the morning, he was again drowsy; in the evening he was seized with convulsions, and shortly afterwards he became comatose and insensible, with dilated pupils and stertorous breathing. He was bled to ℥x, and much relieved thereby. The coma ceased, and consciousness and speech returned in a quarter of an hour. Mustard was applied to the feet, and a draught containing tinct. canthar. and sp. æth. nit. in camphor water was given every third hour, with a view of restoring the accustomed diuresis. On the morning of the 5th he was conscious, and still had some headache. The diuretic mixture was continued, and a black draught administered immediately. On the 6th he was again found in a semi-comatose state, the pupils were dilated, and there was stertor, with sighing respiration. Six leeches were applied to the temples, mustard to the feet, and cold to the head. Then coma became more profound, and he died at 9 P.M.

Autopsy.—The kidneys were found to be reduced to mere sacs, of from twice to thrice the extent of the healthy kidney. There was a complete absence of all proper parenchymatous substance, both tubular and cortical; the sacs being divided into a number of cells by the intertubular septa which occur in the fœtal state. The walls and septa were formed of strong fibrous tissue, lined with what appeared rather serous than mucous membrane, and the cavity and ureters contained a small quantity of the same urinous fluid which had been passed during life. The ureters were so much dilated that that on the right side was at first mistaken for the ascending colon. The circumference of the ureter varied from three to four and a half inches. The kidney and ureter of either side were almost precisely in the same condition. The urine in the ureters and sacs was tested for urea by evaporation and nitric acid, without result. On closer examination no proper kidney substance could be discovered, nor did it appear that there ever had been any tubular or cortical portions; here and there were a few hard cartilaginous masses of very small size, closely adherent to the membrane forming the sac. The other abdominal and the thoracic organs were healthy. The brain was not examined.

CASE 5.—On the 29th of May, 1862, I saw, with Mr. J. Smith, of Stretford Road, a youth of 16 years of age, who was passing a large quantity of a watery urine. He was moderately well-grown, exceedingly emaciated, weighing only 78 pounds. Pulse 127; tongue glazed, red in the centre, and covered with a yellowish-brown fur at the sides. The skin was dry and harsh. The patient was troubled with intense and incessant thirst, and voided from nine to twelve pints of urine daily. The appetite was bad. Neither the head nor chest was the seat of any subjective symptom.

He gave the following account of himself: Previous to his present illness he was occupied as a clerk in a warehouse, and had enjoyed uninterrupted health until three months ago. About that time he noticed that he was getting thinner and weaker, that he drank a great deal, and never perspired. These symptoms had undergone a gradual and steady increase, and a fortnight ago had sustained an alarming aggravation. The patient could, nevertheless, still go about, and even take the air for short periods. He suffered no pain in any part, but he slept badly, and passed restless nights. The appetite had been indifferent from the very beginning, and it was now altogether lost. The bowels were moved almost daily, but there was a tendency to constipation. Dyspeptic symptoms—heaviness after food, flatulence, and occasional vomiting—had been noted from the commencement of the illness, but they did not attain a great severity at any time.

In searching back among the patient's antecedents for any determining cause, no fact of moment was elicited. The lad had been living in comfort, well-clad, well-fed, and well-housed, with his grown-up sisters. No tuberculous or other family taint could be traced. The case had been treated with morphia, bismuth, and permanganate of potash, but with no result beyond a palliation of the dyspeptic symptoms.

The urine of the twenty-four hours was carefully collected and measured on six several occasions, and portions sent to me for examination. The characters of it were constant; it was pale like water, and the specific gravity varied from 1002.7 to 1004. The quantity was between nine and ten pints at the time of my visit. It afterwards increased to fourteen pints daily. There was neither albumen nor sugar in it, and its reaction was faintly acid. The quantity drank was found, on exact measurement, to be almost precisely equal to the quantity of urine. The amount of urea varied from 0.4 to 0.55 per cent., and from 394 to 505 grains in the twenty-four hours. This was an enormous quantity for the weight of the body. According to the mean results tabulated by Dr. Parkes, the daily secretion for his weight of 78 pounds should only have been 275 grains.

The patient continued without much change beyond a progressive increase of debility and loss of flesh, drinking enormously, and voiding corresponding quantities of urine, until July 5th, when he was suddenly seized with convulsions and insensibility. After the convulsions had ceased, he began to recover some degree of consciousness, and passed into a semi-comatose condition, which persisted for three days, and then passed away. During the period of unconsciousness the diuresis diminished notably; but it returned immediately afterwards, and the patient continued very much as he was before the seizure, for a period of ten days, when he was again taken with convulsions and insensibility, and died on the morning of July 18th.

Autopsy.—Thirty hours after death. The body was emaciated to the last degree; signs of incipient putrefaction appeared on the abdomen, the weather being warm.

Chest.—The heart was healthy, but very small; the lungs were stuffed with crude tubercle throughout their upper lobes, and several small vomicæ lay scattered through them.

Abdomen.—Five tuberculous ulcers were discovered in the small intestines; some of them had penetrated the mucous and muscular coats, and seemed ready to break through the peritoneum. There was no tubercular deposit in the peritoneum generally, nor any in the liver or spleen.

The *kidneys* were voluminous, smooth, flaccid, and the two together weighed eight ounces. On section they showed no disproportion between the pyramidal and cortical portions, nor any other morbid change. Examined microscopically, the tubes and cells appeared normal.

Head.—About two ounces of clear serum escaped from the arachnoid sac. The meninges were free from tubercle, and quite natural. The ventricles were greatly distended, and contained six ounces of clear serum; their parietes were macerated, and gave way with the slightest traction.

A nodule of yellow tubercle, of the size of a hazel-nut, lay embedded in the left hemisphere, in the border of the longitudinal fissure, midway between its extremities, and cropping out on the surface. Another nodule, as large as a garden-bean, was found in the posterior border of the right half of the cerebellum. An undue vascularity prevailed at a few spots of the surface of the encephalon. Apart from what has been related, the brain substance was healthy and of firm consistence. The floor of the fourth ventricle was especially examined; it was pale and natural, with no tubercular mass in its immediate vicinity.

CASE 6. (Mascarel—"Gaz. d. Hôp.," February 23, 1863.)—The patient was a man, aged 50, pale and thin, without fever, but a devouring thirst, and a red tongue; appetite good, but not voracious. He drank daily from eight to ten pints of water, and voided urine proportionally. The disease had existed eight months. Seven days after entering the hospital, he became feverish, at first only in the night, then continuously, with nausea, and epigastric tenderness. Thirst was intense, but there was no appetite. Not the least trace of sugar or albumen existed in the urine. The urine showed, after the fever became persistent, on cooling, a slight yellowish-white deposit, not mucous, but as if purulent. This last character was only noticed two days before death.

Autopsy.—The left kidney was more voluminous than the right, and eight to ten little abscesses, varying from the size of a pin's head to a small filbert, were found in the cortical part. The smaller abscesses contained almost concrete pus, and the larger ones fluid pus, without any tubercle. The infundibula were filled with a creamy fluid. All the abscesses were near to and reached the surface.

The right kidney was of natural size, hyperæmic and free from disseminated abscesses, but a lactescent fluid could be squeezed from the pyramidal portions.

The brain was not examined.

CASE 7. ("Revue d. Hôpitaux," 1861.)—A man, æt. 35, who had had saccharine urine five years before, was passing daily six to seven litres of urine, sp. gr. 1001–7, not containing either albumen or sugar. The patient was suffering from phthisis. Being seized with acute pulmonary symptoms, the urine fell to a small quantity; a purpuric eruption came out on the skin, and death took place fourteen days after, without cerebral complications.

Autopsy.—The walls of the fourth ventricle were more vascular than usual, and some tawny spots were seen disseminated on the surface. On making transverse sections of the spots, Luys discovered with the microscope extensive fatty degeneration of the nerve cells.

CASES 8 and 9. (Kien—"Gaz. Hebd.," 1866.)—In one case Kien found great vascularity of the kidneys. In the walls of the fourth ventricle, scattered yellow spots were found, which exhibited under the microscope abundant evidence of fatty degeneration of the nerve elements.

In his second case, no changes were found in the kidneys—neither with the naked eye nor with the microscope, beyond congestion of the Malpighian bodies. Nothing was found in the medulla oblongata.

CASE 10. (Reported by Mosler in Virchow's "Archiv," 43, 225.)—The patient was a girl of 22, who had for many years been suffering from symptoms of tumor of the brain. In the later years of her life she was, in addition, suffering from symptoms of diabetes insipidus. After death, a tumor as large as a walnut, of a fibro-plastic nature (Gliosarcoma of Virchow), was found attached to the floor of the fourth ventricle, and filling the entire cavity.

CASE 11. (Dickinson on "Diab.," p. 184.)—A child of 5 had symptoms of D. insip. for about a year. She died with symptoms of tubercular meningitis. Miliary tubercles and thickening were found at the base of the brain near (but not in) the fourth ventricle, and on the upper surface of the cerebellum; no bulky masses of tubercle were found.

CASE 12. (Dickinson on "Diab.," p. 223.)—A man, æt. 60, had suffered from diabetes insipidus for fifteen months, and had been ailing for two months before the polyuria was noticed. Post-mortem there was found malignant disease of the liver and post-peritoneal glands, which had involved and partially destroyed the solar plexus. The kidneys were of normal size, but minutely injected. Microscopically there was found some excessive growth of the tubular epithelium.

CASE 13. (Ralfe, "Lancet," 1881, I. p. 406.)—A man, æt. 24, with a syphilitic history, had suffered from polyuria with increase in the amount of phosphates excreted. Post-mortem there was found a gumma about the size of a small hazel-nut, situated under the floor of the third ventricle in the middle line. It was surrounded by some softening of the cerebral substance.

CASE 14. (Haas, "Prager-Vierteljahresch.," 1875, Bd. 127, p. 12.)—A woman, æt. 23, suffered from polyuria, associated with almost complete blindness and tuberculosis. The urine passed varied from five to ten litres a day, and occasionally contained a little albumen. There was found post-mortem tuberculosis of the lungs and intestines. The optic nerves were atrophied, but no change of importance was found in the brain. The kidneys were not enlarged, but were congested. The liver was enlarged and hyperæmic.

CASE 15. (Fazio—*see* "Lond. Med. Record," 1880, p. 138.)—A woman, aged 21, had suffered for three years from excessive thirst and polyuria, with pains in the head. Dimness of vision was noticed, but no ophthalmoscopic examination was made. At the autopsy a sarcomatous tumor was found in the region of the sella turcica, causing pressure on the brain substance in the neighborhood.

NATURE OF DIABETES INSIPIDUS.—A review of the *post-mortem* examinations just recorded, is sufficient to show that the initial disorder in diabetes insipidus must be looked for elsewhere than in the kidneys. The diverse organic alterations found in the kidneys by Eade, Neuffer, and Mascarel, were evidently secondary, and produced by the irritation of the frequent micturition and excessive and long-continued diuresis. Similar alterations are found in the kidneys of persons dying of long-standing saccharine diabetes. The case of Dr. Strange is certainly very puzzling: one can only conceive a teleological reason for the diuresis, namely, the absolute necessity for an immense transudation of watery fluid to make up for the imperfection of the glandular apparatus.

Nor can the disease be regarded merely as excessive thirst and a vicious habit of profuse potation. It has been almost invariably found that an enforced diminution of liquids fails to arrest the diuresis, except partially. The observations of Falck, Neuschler, and Neuffer, agree perfectly in this: that when the supply of water by the mouth is diminished, the quantity of urine notable exceeds the ingoing water, and thereby occasions dehydration of the tissues, with an intolerable aggravation of the symptoms.

It may be regarded as probable that the *immediate* anatomical cause of polyuria is a dilation of the renal capillaries, whereby their walls are thinned and rendered favorable to increased transudation of watery fluid from the blood. But how is this brought about? It is now generally believed that the minute bloodvessels possess in their circular and longitudinal muscular coats a provision for an active expansion as well as an active constriction of their calibre.[1] This provision is under the control of the sympathetic branches of nerves (nervi vasi-motores), and serves to maintain the aqueousness of the blood within certain limits of health. When the tissues and blood are overcharged with water, the renal vessels expand, and permit a copious transudation of an aqueous urine; when, on the other hand, the system is undercharged with water, they contract, and thereby restrict the urinary transudation. In diabetes insipidus this endowment seems greatly impaired; the renal capillaries appear to resemble the iris in glaucoma, which remains in a motionless, semi-dilated state, and neither contracts with light nor dilates with belladonna. In polyuric subjects the contractile power of the renal vessels is apparently paralyzed; and the power of regulating the urinary flow consequently lost. If a healthy person imbibe an excessive amount of water, he rapidly gets rid of the overplus by a sudden and copious diuresis, and then the secretion falls quickly to its ordinary rate: but a polyuric subject, under similar conditions, shows very little *immediate* increase of urine, but a steady, persistent, though less intense, augmentation, lasting many hours, and which is not succeeded by a fall to the ordinary standard. On the other hand, if a healthy person imbibe a lessened quantity of water, the discharge of urine falls in proportion: whereas the polyuric, under the same circumstances, shows no such adaptation; he still continues to discharge an undue amount of urine, which necessitates constant imbibition of new supplies of water to prevent dehydration of the tissues.

On this view, the primary cause of diabetes insipidus must be looked for in some other parts than in the kidneys; namely, in some part of the chain of sympathetic nerves which controls the action of the contractile tissues of the renal vessels. This chain extends from the kidneys to the abdominal ganglia, thence to the spinal cord and the floor of the fourth ventricle, where the sympathetic system seems to have its centre. From above, this centre receives impressions from the encephalon.

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[1] For a demonstration of the anatomical possibility of this endowment I must refer to Schiff's ingenious researches. *See* his "Untersuchungen über die Zuckerbildung in der Leber," p. 92. Würtzburg, 1859.

This theory seems conformable both to experiment and to clinical facts. Bernard found that by puncturing a certain spot in the floor of the fourth ventricle, an augmented secretion could be produced of a watery urine, containing neither sugar nor albumen. A large proportion of the cases of diabetes insipidus followed injuries to the nervous centres, or were evidently dependent on some derangement of the nervous system. In six cases, palpable disease of the brain was found after death, while the kidneys were healthy.

Flatten ("Arch. f. Psychiatrie," XIII. p. 671, 1882) has recorded a case which assists to localize the "polyuric" centre. A patient, after a blow on the head, was attacked by diabetes insipidus. There were also certain permanent symptoms of the cerebral disturbance. Thus, there was paralysis of the left sixth nerve and weakness of the right sixth, with slight deafness on the left side. The probable diagnosis was hemorrhagic softening near the centres for the sixth nerves. Flatten calls attention to two other cases, one reported by Gayet ("Gazette Hebdomad.," No. 17, 1876), in which paralysis of the right sixth nerve was accompanied by diabetes insipidus; and another, by Kämutz ("Arch. f. Heilkunde," 1873), in which an injury to the head was followed by paralysis of the right sixth nerve and diabetes mellitus.

It is, perhaps, worthy of remark in this connection that puncture of the floor of the fourth ventricle, near the nucleus of the trigeminus, has produced increased flow of saliva, while in two cases (Külz and Worm Müller) salivation was observed in diabetes insipidus.

In other cases, it is probable that the sympathetic in the abdomen was the point originally injured.[1] Among such may be classed those arising from drinking cold fluids while the body was heated, and perhaps also those following alcoholic excesses.

The influence of a lesion of the vagus in producing the disorder is doubtful. Dr. Ralfe ("Lancet," 1876, I. p. 308) has recorded two cases of aortic aneurism accompanied by diabetes insipidus, and he was inclined to attribute the latter symptom to pressure on the vagus in the chest, on the ground that Bernard produced polyuria by irritating the vagus. Further observations, however, are required to support this view.

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[1] In reference to this point, see two papers by Eckhardt and Knoll, in Eckhardt's Beiträge, VI. Heft i.—also Merbach's paper (Küchenmeister's Zeitsch., 1865, p. 10). Probably the case related by Professor Houghton was of this character. This was a woman who died with symptoms of D. Insipidus, together with distention of the abdomen from fecal accumulation caused by a uterine tumor pressing on the rectum. The mesenteric glands connected with the colon were enlarged and indurated. The symptoms had lasted nine years. Dublin Quarterly, Nov. 1863. See Dr. Dickinson's case mentioned on p. 235.

A feature of the disease favorable to the theory of its nervous origin, is its occasional sudden onset after events which do not directly implicate the urinary organs; and its equally sudden subsidence when a strong impression is made on the system by an intercurrent inflammation. The total and unexpected disappearance of the disease, after continuing many months or years, is more in accordance with the habit of neuroses or nervous diseases than of any other class of maladies.

THE DIAGNOSIS of diabetes insipidus lies on the surface. A permanent increase of the urine, without sugar or albumen, suffices at once to define and to identify it. But it is evident from the facts and considerations before adduced, that to gain a useful clew for treatment, we must attain to more precise notions as to the part originally affected—whether brain, or cord, or abdominal ganglia, and also as to the nature of the lesion in the affected part.

THE PROGNOSIS is, speaking generally, less serious than in saccharine diabetes; nevertheless, insipid diabetes is a very unmanageable complaint; it generally resists treatment, and not unfrequently runs a fatal course. The gravity of the prognosis in a particular case depends on the severity of the general symptoms, and on the presence or absence of complications. The cases which affect the general health the least, though mostly proving incurable, appear to be those which arise after inflammatory complaints, after mental emotion, cerebral injuries, and those which arise early in life without any known cause. On the other hand, those which depend on organic disease of the nervous centres are necessarily fatal.

TREATMENT.—Until we obtain a better insight into the pathology of these cases, our treatment must be necessarily empirical. Hitherto the indications pursued have been mostly confined to efforts to subdue the more palpable symptoms—the thirst and diuresis. The means used for this purpose have been various. J. Frank considered nitrate of potash in large doses as a specific; in some of the recorded cases it proved of decided service; in others it as completely failed. Camphor and valerian were used in a number of the French cases, and sometimes with success. Trousseau speaks in high terms of valerian, and cites the authority of Rayer as additional evidence of its efficacy. Trousseau gave it in large doses. In one case, which ended in complete and permanent recovery in four months, the extract was gradually pushed to the enormous dose of one ounce daily; his ordinary dose would appear to be two and a half drachms a day. Rayer obtained rapid success in a boy who suffered from polyuria, with emaciation and nervous symptoms, by means of the powder of valerian. In the case of the boy alluded to at p. 233, the valerianate of zinc appeared to produce a good effect. It was given in pill, in gradually increasing doses, until 20 grains a day had been reached. The urine fell from 15 to 5 pints a day, and the thirst and dryness of the tongue were greatly diminished.

Enforced abstinence from fluids was tried in a number of cases; and, in one recorded by Becquerel, with good effect; but in nearly all the others it was not only unsuccessful, but was followed by decided aggravation of the general suffering, and in some cases by symptoms of threatening or actual uræmic poisoning. The fate of Dr. Strange's patient is particularly instructive on this point. In one of my own cases opium produced great diminution of the thirst and diuresis, but the patient's distress was so increased that I was compelled to suspend the use of the remedy.

Dr. Murrell[1] obtained good results from the administration of belladonna and ergot, in a case where the disease followed a fall on the head. He believed that here there was a lesion of the sympathetic and consequent relaxation of the renal vessels, which would probably be overcome by the drugs administered. In other hands, also, ergot has met with considerable success.

Among other remedies occasionally followed by success were iron, gall-nuts, lime water, cream of tartar, iodide of mercury, iodide of potassium, and pilocarpin. Dr. Kennedy ("Practitioner," vol. 20, p. 94) has recorded five cases in which great benefit was obtained from drachm doses of dilute nitro-muriatic acid.

One of the most frequent incidents in the history of diabetes insipidus is the temporary suspension of the thirst and diuresis on the occurrence of some intercurrent febrile affection, and in two instances the suspension proved permanent. A hint for treatment may be taken from this. The application of a large blister on the nape of the neck or the epigastrium (according as the associated symptoms and the anamnesis point to the nervous or the digestive system), might in some cases have the same beneficial effect as a spontaneous inflammation. In the case treated with opium, just alluded to, a blister to the pit of the stomach proved of more benefit than any of the numerous means previously employed.

The application of the constant galvanic current has been tried, with a promise of success. Dr. M. Seidel tried this treatment in a woman, æt. 29, suffering from diabetes insipidus. He applied one pole of a strong battery over the loins near the spine, and pressed the other pole as deeply as possible upon the corresponding hypochondrium; each side was daily galvanized for 5 minutes. In 8 days the urine had fallen from 5957 c. c. to 4600 c. c., and after three weeks to 2300 c. c., and next month to 1904 c. c. Simultaneously the weight of the body increased by nine pounds. The amendment was found to be maintained at the end of three months.[1] Külz obtained very favorable results in two cases treated for some weeks by a constant battery of 30 to 40 elements. He applied one pole as high as possible to the nape of the neck and the other to the loins or epigastrium. The most effective way appeared to be to apply the positive pole to the nape and the negative pole first to the loins for four minutes, and then to the pit of the stomach for four minutes. Dr. Althaus[2] has recorded a case in which immediate improvement resulted from the application of the constant current. The current was applied to the occiput, its direction being repeatedly reversed.

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[1] Brit. Med. Journ., i. 1876.

The secondary symptoms—dryness of the skin, epigastric and lumbar pains, etc., must be treated by warm baths, alkaline tonic infusions, sedative and anodyne remedies.

APPENDIX.

Cases characterized by excessive diuresis and thirst; urine of very low specific gravity, but containing, or having contained, a trace of sugar.

Cases of this type form an intermediate group between insipid and saccharine diabetes; and their existence completes, in an exquisite manner, the correspondence between the results obtained by Bernard from artificial injuries to different parts of the floor of the fourth ventricle, and clinical observations.

Two cases of this class, following fracture of the skull, are reported by Fischer ("Archives Gén.," Oct. 1852). In one, the sugar amounted to 0.32 per cent. In the other, in which there was a voracious appetite as well as intense thirst, there was 0.5 per cent. on the first day after the accident, and 0.6 per cent. on the third day. The floor of the fourth ventricle was examined in both instances after death, and was alleged to be healthy; in the second case (which terminated in tetanus), the whole brain and cerebellum, so far as could be made out, were uninjured.

A third case, arising spontaneously, is related by Trousseau. The disease had already existed four years without serious giving way of the health. The examination of the urine (by

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[1] Schmidt's Jahrb., Bd. 130, S. 97.

[2] Med. Times, 1880, ii. p. 617.

Bouchardat) on two occasions, at considerable intervals of time, showed a trace of sugar. The quantity of urine varied from 12 to 37 litres a day. Among the secondary symptoms were impotence, lumbar pains, and a remarkable tolerance of alcoholic drinks. This man derived considerable benefit, but was not cured, under the use of valerian.

A fourth case is recorded in the "Gaz. des Hôpitaux" for 1861. A man, æt. 35, was afflicted for many years with polyuria, passing daily from 10 to 12 pints of urine, specific gravity 1001–1007. He was the subject of chronic phthisis when in the Hôtel Dieu, under Trousseau, in 1861. There was then not a particle of sugar or albumen in the urine; but when he was an inmate of the Hôp. St. Antoine, in 1856, a trace of sugar was found. Acute pulmonary symptoms came on at last, with purpura. The urine rapidly diminished in quantity, and the patient sank. The autopsy was performed by Luys. The floor of the fourth ventricle was more vascular than natural; large vascular trunks mapped the surface; yellow spots were seen scattered over the upper part, near the crura cerebri. Similar patches were found below the points of origin of the radicles of the portio mollis. On section, the whole gray substance was found unusually vascular, and of a rosy hue. Microscopic examination showed that these alterations in color were due to fatty degeneration of all the nerve cells of the corresponding regions.

A fifth case is reported by Dr. Ralfe ("Lancet," 1881, i. p. 407). A gentleman, æt. 37, had suffered from mild glycosuria for eighteen months. The glycosuria occasionally disappeared, but a certain amount of polyuria (2500 c. c. daily) with excessive excretion of phosphates was constant. The patient improved under codeia and opium, with tepid saline douches.

A case which may be classed with these occurred to myself some years ago. A man of sixty-five was brought into the Manchester Infirmary in an apoplectic fit. He died after lying for six hours in deep coma. During this period he flooded the bed with urine. After death a large quantity of urine was withdrawn from the bladder. It had a specific gravity of 1010, and contained a considerable quantity of sugar. A voluminous clot was found in the brain.

CHAPTER II.

DIABETES MELLITUS.

THE multiplied researches of recent years on the occurrence of sugar or glucose in the urine, necessitate the adoption of some classification of cases of saccharine urine.

Cases of saccharine urine may be primarily divided into two broad classes or divisions.

One class consists of instances in which a small quantity of sugar appears in the urine for very short periods, without relevant symptoms—the circumstance being a temporary and incidental consequence of some physiological or pathological antecedent which has little or no affinity to diabetes, as clinically understood. Belonging to this class are examples of saccharine urine after the administration of chloroform, after eating an excessive quantity of saccharine and amylaceous food, in recovery from cholera, and after a paroxysm of whooping-cough, asthma, or epilepsy. These may be designated as cases of *incidental glycosuria*.

In the other class of cases the glycosuria is more intense; it constitutes a permanent symptom, and persists for considerable periods of time, and is associated with a serious departure from health. To this class alone is the term *diabetes* at all applicable.

This second class again is divisible into two groups. In the first, the glycosuria is persistent and intense, and the flow of urine is greatly increased; this state of urine is associated with thirst, debility, emaciation, and a train of grave, fatally tending symptoms, which constitute a familiar, easily recognized clinical unity. This is the classical *diabetes* of authors, and to this the name diabetes was limited, before our more refined and ready analysis disclosed the presence of sugar in the urine in a number of other and different states.

The second group embraces those less serious types in which sugar is present in the urine, sometimes abundantly, sometimes scantily, sometimes persistently, sometimes intermittently; always with a weakly condition of health, but without thirst or conspicuous emaciation, often, indeed, with corpulence; without any, or only slight, increase in the quantity of urine, and without that fixed tendency to death which stamps the first group—occurring also generally in advanced years, or at least beyond the time of early manhood. Some of these *milder* types of glycosuria will be separately noticed at the end of the present chapter.

ETIOLOGY OF DIABETES MELLITUS.

In the decade 1851–60, 4546 deaths from diabetes were registered in England and Wales, being an annual average of 454.[1] Of the total number 3032 were males, and 1514 females, showing that in this country diabetes is twice more common in men than women. Up to the age of puberty, the two sexes appear to be equally liable to diabetes; but from that period on to old age the liability of the male sex maintains an increasing ratio, as may be seen from the following table:

TABLE *showing the number of deaths from diabetes, at different periods of life in the two sexes.*[2]

PERIOD OF LIFE.	Under 5 yrs.	5–10 yrs.	10–15 yrs.	15–25 yrs.	25–35 yrs.	35–45 yrs.	45–55 yrs.	55–65 yrs.	65–75 yrs.	75 years and upwards.	All ages.
Deaths in males.	28	40	97	378	468	502	550	500	364	105	3032
Deaths in females	23	42	78	220	282	261	247	191	144	26	1514
Total males and females.	51	82	175	598	750	763	797	691	508	131	4546

Dr. Dickinson has shown that diabetes is more common in agricultural than in urban districts, and is also more frequent in the colder parts of the country.

Diabetes prevails chiefly among young and middle-aged adults. It is rare under five years of age. The youngest example that has come under my notice was a boy of three years; but in the Registrar-General's "Reports" for 1851–60, ten deaths from diabetes under the age of one year are registered, and as many as thirty-two under the age of three years. The mortality from diabetes attains its maximum between the ages of twenty-five and sixty-five years, and maintains itself between these epochs with tolerable uniformity. In extreme old age deaths from diabetes are more rare, not only absolutely, but as compared to the mortality from all causes.

The development and exercise of the *sexual functions* appear to have a marked effect in increasing the liability to diabetes in both sexes; and the diminished frequency of the disease in women after the age of forty-five (as compared with men) corresponds with the earlier decline of the sexual activity in the female sex. The maximum mortality in males is between forty-five and fifty-five years; in females between twenty-five and thirty-five years.

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[1] In the eight years 1861–8 the annual average of deaths from diabetes was 628.

[2] Constructed from the Registrar-General's Reports, for 1851–60, for England and Wales. Mean population for the decade, 19,000,000.

Dr. Mathews Duncan has pointed out that while a slight glycosuria is common in pregnant women, sometimes true diabetes supervenes, and is then very dangerous to the life of both mother and child ("Lancet," 1882, ii. p. 944).

HEREDITARY INFLUENCE is a distinct predisposing cause of diabetes; and I have known a considerable number of instances in which the disease has run in families. Seegen mentions a brother and sister who were both diabetic. A short time ago I had under my care an uncle and niece similarly affected. I have also a note of a family of eight children, all of whom became diabetic, though the parents were healthy. Sir H. Marsh[1] refers to a family in which diabetes could be traced to the second generation, and to another family in which it could be traced through four generations. Schmidtz, of Neuenahar, traced heredity in 248 out of 600 cases (*see* "Lancet," i., 1883). Diabetic patients have frequently been observed to belong to families in which phthisis and epilepsy prevailed. One of my patients was one of four survivors out of a family of twenty-five; twenty of these had died, after reaching adult age, of lingering complaints with great emaciation, probably phthisis or diabetes.

The subjects of obesity and of the gouty diathesis are very prone to a mild form of diabetes.[2]

EXCITING CAUSES.—The exciting cause of diabetes is often obscure. In a considerable number of cases the disease has broken out soon after exposure to *wet and cold;* in others after copious drinking of cold fluids while the body was in a heated state. Cases arising from cold, generally present a train of neuralgic or *quasi* rheumatic symptoms, before the breaking out of the thirst and diuresis. Excessive use of *saccharine and amylaceous* articles of food, antecedent *acute febrile diseases*, abuse of *alcoholic* drinks, have all been noted as probable exciting causes of diabetes.

The disease is sometimes traced to a violent *mental emotion.* In one of my patients it followed on distress of mind caused by an unjust suspicion of theft; in another it followed the burning down of his place of business; in a third it was attributed to anxiety attendant on a Chancery suit. Rayer mentions a case of diabetes coming on after a violent fit of anger, and Landouzy another after violent grief. Dr. Dickinson cites several striking examples of diabetes following excessive mental anxiety.

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[1] Dublin Quarterly, 1854, p. 17, note.

[2] From a discussion in the Academy of Medicine of Paris, it would appear that glycosuria is very common among those who have suffered from malaria. *See* Hayem's Revue, 1883, ii. p. 164 Sir J. Fayrer's experience also confirms this opinion. (Brit. Med. Journ., 1882, i. p. 529.)

Organic diseases of the brain and cord have, of late years, been shown to be occasionally the exciting cause of diabetes. These cases are of special interest as bearing on the discoveries of Bernard and others, on the artificial production of glycosuria in animals by cutting or puncturing various parts of the nervous system.

On Feb. 19, 1871, I saw, with Dr. Ransome, of Bowdon, a contractor, aged 55. He had been formerly in the habit of taking spirits freely—but three years ago he had a succession of epileptic fits, in consequence of which he was induced to take light claret only. The fits then ceased. For the last twelve months he had taken largely of milk—sometimes four or five quarts daily. He came under Dr. Ransome's treatment in Jan. 1871, for loss of memory and mental confusion. He was a stout, florid man—though somewhat thinner than he had been. There was much thirst and a ravenous appetite. The pulse varied from 120 to 140. The urine amounted to between 10 and 12 pints daily, and was highly saccharine. By strict dieting it was reduced to 7 pints, with a sp. gr. of 1032–40. On June 1st he had a slight paralytic seizure affecting the right hand and arm—with loss of speech for a few minutes. His loss of memory was complete for recent events—even a few minutes after their occurrence—but he retained full recollection of matters which had occurred before his illness. This man died some months later. A naked-eye examination of the brain did not reveal any palpable signs of disease.

Pavy cites the following examples: A late alderman of the city of London was seized with cerebral hemiplegia. His urine was tested by Dr. Barlow at the period of the attack and found to be free from sugar. There had also been nothing from the symptoms and history to lead to the suspicion that sugar would be found. Shortly afterwards, however, strongly marked diabetes set in. A member of the medical profession, who was seen by Sir W. Gull, was seized at the age of fifty-two with an apoplectic fit, from which he recovered, with hemiplegia, however, of the left side of the body remaining behind. Five weeks after the fit, this person who had never previously presented any symptoms of diabetes, began rapidly to emaciate, which led to an examination of the urine being made. A highly saccharine state of it was found to exist.

Dr. Richardson[1] relates a case of diabetes in which convulsions and symptoms of meningitis occurred during life: after death an ossific growth was found pressing on the pons Varolii, and an abscess in the posterior cerebral lobes. Dompeling relates a case of diabetes in which a tumor as large as a nut was found after death, occupying the whole of the right half of the medulla oblongata ("Arch. Gén.," May, 1869).

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[1] Med. Times and Gaz., March and May, 1862.

E. Fritz has collected an interesting series of cases of diabetes associated with various organic diseases of the brain and cord (cerebral softening, tumors of the pia mater, general paralysis, and myelitis).

Mr. Morison has reported a case of glycosuria accompanied by organic disease of the brain.[1] The patient was a man aged 49 years, who had a history of syphilis, and had also suffered from nephritic colic. Five years before death he had complained of aphasia, and paresis of the right side, from which he recovered in a few days, but since that time had urinated excessively. About two years and a half before death he was found to be suffering from hemianæsthesia of the left side, and progressive paresis of the same side. The patient in addition showed the symptoms of glycosuria rather than of true diabetes. Post-mortem, there were found çysts in the right optic thalamus, and a granular condition of the lower three-fourths of the fourth ventricle.

Dę Jonge reports a case in the "Archiv f. Psychiatrie," xiii. p. 658, in which a tuberculous tumor was found in the medulla of a patient, who during life had suffered from diabetes.

Still more numerous are the cases in which diabetes has followed *external injuries* to the brain and other parts. Dr. Goolden[2] has published a series of such examples; and P. Fischer has increased the list to twenty-one cases, and ably analyzed them. The injuries consisted of blows and falls on the forehead, vertex, and occiput—sometimes with and sometimes without fracture of the skull. In some instances there was temporary loss of consciousness, in others not.

In addition to cases of violence directly affecting the brain, a large number (twenty-two cases) are cited by Fischer of blows on the face, fractures of the vertebræ, blows on the loins, the thorax, the abdomen, contusions of the kidney and liver, violent efforts, etc., which have been followed by diabetic symptoms.

Some of these traumatic cases were examples of confirmed diabetes of the ordinary type, and ran a fatal course. Others were of much milder type, transitory in their duration, passing away on the subsidence of the cerebral symptoms. In some of them the diabetic symptoms commenced at the time of the accident, or shortly after; in others, not until some months had elapsed after the injury.

In a certain proportion of the traumatic cases (eight out of forty-three collected by Fischer) the urinary disorder consisted of simple polyuria.

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[1] Ed. Med. Journ., March, 1878.

[2] Lancet, June and July, 1854, and Med. Times, Dec. 1854.

It is probable that in all the traumatic cases the injury (however different its seat) implicated some part of the sympathetic nervous system, either within the cranium or spinal cord, or in its peripheral distribution.

Lesions of the peripheral nervous system may give rise to glycosuria, and occasionally it is said to diabetes, probably by reflex action. Neuralgias of the fifth pair of nerves, and of the sciatic nerves, seem specially prone to produce this condition.

In his recent treatise on diabetes, Frerichs lays great stress on the influence of lesions of the nervous system in producing the disease. Out of 165 cases, he was able to trace some form of nervous lesion in 75. The nervous disturbances comprised organic lesions of the brain, mental disorders, affections of the peripheral nerves, concussion of the brain, and excessive mental strain, trouble, and worry.

SYMPTOMS.

The invasion of diabetes is generally insidious. The disease is seldom recognized until it has existed some weeks or months. The initial symptoms (malaise and slight emaciation) pass unnoticed, because the appetite continues good; but the patient's suspicions are at length aroused by the increasing frequency of the calls to make water, and an incommodious thirst. The disease sometimes, however, attains a high intensity in a few weeks. In one of my cases as much as fifteen pints of urine a day were secreted in the third week.

As the disease advances, it assumes its characteristic features. The thirst becomes insatiable, the appetite excessive or voracious, the skin harsh, dry, and scurfy; the patient emaciates; the countenance wears an appearance of suffering, and the features are drawn; a distressing sinking is felt at the pit of the stomach; the tongue is glazed and furrowed; a scanty, tenacious mucus gathers in the mouth, which is parched and clammy; the urine rises to eight, twelve, or more pints in the twenty-four hours; this urine is of a pale straw color; its density varies from 1035 to 1045 or higher; and it contains a large proportion of sugar; the virile powers fail; and the mental faculties lose their wonted vigor.

If the malady proceeds unchecked, these symptoms increase in intensity. The emaciation and loss of strength attain an extreme degree; pulmonary symptoms resembling those of phthisis often make their appearance, and advance with alarming rapidity; or colliquative diarrhœa sets in; hectic fever is established; the urine now diminishes in quantity, perhaps loses its sugar and becomes albuminous; the legs become œdematous and the unfortunate sufferer is at length released, often very suddenly, either by sheer exhaustion, or he is carried off by one of the numerous complications of the disease.

Some of these symptoms require a more detailed consideration.

THE URINE.—The essential features of the urine in diabetes are its excessive quantity and the presence of sugar. The proportion of the latter varies from 8 to 12 per cent. It is chemically identical with grape-sugar or glucose. The quantity of sugar excreted daily ranges from 15 to 25 ounces; but it may amount to two pounds or more, or fall to an ounce or less. The proportion of sugar is always increased after food, and diminished after fasting. After the use of starchy or saccharine substances, the increase is much greater than after animal food. In many of the milder cases, and probably in the earliest stages of all, the urine becomes free from sugar when starch and sugar are entirely withdrawn from the diet; but in confirmed cases the urine still continues saccharine—though in greatly diminished intensity—when the diet is purely animal, and even when no food at all is taken. In this last case the sources of the sugar are necessarily the tissues of the body.

The density of diabetic urine usually fluctuates a few degrees above or below 1040; it may rise to 1055 or 1060, or sink to 1015.

Intercurrent inflammatory or febrile attacks cause the sugar to diminish, or even temporarily to disappear. Toward the approach of death a similar diminution is observed.

The quantity of the urine oscillates usually between 8 and 15 pints daily; but it has been known to exceed 32 pints. When the diet is restricted to animal food, the urine is generally reduced to four or five pints a day. The quantity of the urine is about equal to the liquids imbibed. The opinions formerly held, that the urine exceeded the ingested liquids, and that the body absorbed water through the lungs and skin, or generated it *de novo* from the elements of the food and tissues, are quite unsupported by more rigid observations of recent date.

When the flow is considerable the urine has a very pale straw tint and a peculiarly bright aspect. It speedily becomes opalescent when exposed to warm air, and in a few hours ferments with abundant disengagement of gas and production of sporules and filaments of the yeast plant. These latter form a white flour-like deposit in diabetic urine after it has been kept awhile.

When the flow does not exceed four or five pints, the color and general appearance of the urine are natural.

With regard to the ordinary constituents of the urine, no particular alteration takes place in their rate of excretion beyond a diminution of their proportion to the water, and (generally) some absolute increase of their quantity. Very contradictory statements have appeared on this point; but the more trustworthy observations appear to support the above conclusion, especially with respect to urea. Uric acid is often difficult to detect, owing to the immense proportion of water; but it is not really absent, as has been alleged; and when the volume of the urine is reduced to four or five pints a day, uric acid frequently forms an abundant deposit of large dark red crystals. More rarely I have observed oxalate of lime; and in one instance a persistent deposit of crystallized phosphate of lime.

The presence of a small quantity of albumen, and even of blood, is not uncommon in advanced cases, and constitutes an untoward sign. In a gouty old gentleman who was passing four pints of a moderately saccharine urine a day, I detected (in addition to a little albumen) transparent fibrinous casts of the uriniferous tubes.

Thirst is one of the earliest and most persistent symptoms of diabetes, and has often led to its detection. Diabetic patients will generally drink from 8 to 12 pints a day; but sometimes they imbibe as much as 25 or 35 pints a day. Nevertheless, this enormous potation does not suffice to quench the intolerable thirst, nay, it seems even to intensify it. A perpetual painful dryness of the mouth and fauces remains in spite of a deluge of water. Patients have even been known to drink their own urine to allay their craving for fluids.

The immediate cause of this thirst is, probably, the existence of sugar in the blood. This crystalline substance, like any other crystalloid, creates a demand for water to effect its dissolution and elimination from the body. On the other hand, the consumption of large quantities of water seems to aggravate the disease, by accelerating the disintegration of the tissues.

Inordinate *appetite* is not nearly so constant a symptom as excessive thirst; and in the course of the complaint there is not unfrequently complete anorexia. Toward the fatal termination a loathing for food of every sort often prevails, and is accompanied by rapid sinking of the powers of life.

Emaciation is generally a prominent symptom; and the degree of it is proportional to the intensity and duration of the disease. The disappearance of fat is probably not without direct connection with the unnatural transformation of the amyloid substance of the liver into sugar; as it seems highly probable that the normal destination of this is, partly at least, to nourish the adipose tissues of the body. Emaciation is, however, not an invariable concomitant of diabetes. One of my patients weighed over 15 stone, though he had been voiding daily 12 pints of highly saccharine urine for some months. One of Prout's diabetic patients weighed 23 stone! This obese class of cases is markedly less severe, and of more hopeful prognosis than the generality.

The emaciation is not confined to the fatty tissues; the muscles become atrophied, and even the heart itself. The enormous flow of fluid through the kidneys explains to some extent the excessive emaciation of diabetic patients; for it has been shown by Genth, Böcker, and Mosler, that simple transudation of water through the body increases the disintegration of the tissues, and induces rapid loss of weight, unless the deficiency be made up by increased supplies of food. In diabetes, notwithstanding the enormous amount of aliments ingested, the defective state of the digestive organs prevents the possibility of suitable compensation by increasing the supply from without. In agreement with this view, it is found that when the flow of urine in diabetic patients is brought down by appropriate treatment to three or four pints à day there is usually no further loss of weight, or even the patient recovers some of what he has lost, though the urine still continue saccharine.

Dryness of the *skin* is a usual and very unpleasant symptom of diabetes, and its intensity is proportional to the diuresis. Some diabetic patients, however, sweat freely throughout their complaint; others only begin to sweat on the advent of hectic fever.

The temperature of the body is usually normal—often sinking a little below the normal—sometimes even markedly so. Even in intercurrent inflammations it scarcely rises above the healthy standard.

The prevalence of *boils* is a curious occasional coincidence of saccharine urine. In a gentleman recently under my care, successive crops of boils were the earliest symptom of the disease. Sometimes they constitute veritable furunculi, and as many as twenty-two have been counted at the same time on a diabetic patient. They may even be the immediate cause of death. Other cutaneous affections are sometimes seen, less frequently than boils, namely, general eczema, psoriasis, and impetigo.

An erythematous and excoriated condition of the urethral orifice (due to the irritation of the saccharine urine) is occasionally a source of great discomfort; and in the female, heat and itching about the vulva are common and distressing symptoms.

Dr. Garron states that *œdema of the legs* is a constant feature in diabetes. It certainly is very common; and the flat surfaces of the tibiæ can nearly always be made to pit on firm pressure, even when no fulness exists about the ankles. I am satisfied, however, that this pitting, when very slight, is not due to œdema, but rather to the soft atonic state of the subcutaneous tissues.

Ascites and *œdema of the arms and hands* occurred in one of my cases. Ascites is also mentioned in one case by Fischer, where the disease was complicated with cataract.

The dryness of the *mouth* usually corresponds to that of the general surface. The tongue is commonly red, preternaturally clean, cracked, and denuded of epithelium. In the less severe cases, or when amelioration has been brought about by treatment, the tongue is moist, and coated with a thin yellowish-white fur. In the majority of diabetic patients the *teeth* are gradually destroyed by caries; and the gums become spongy, swollen, loosened from the teeth, and liable to bleed. The destruction of the teeth is attributed by Falck to the excessive acidity of the saliva, due to the generation of lactic acid from the sugar present in the secretions of the mouth.[1] Sometimes, however, the teeth are preserved perfectly in persons who have been diabetic for many years.

The *digestive organs* rarely bear the unnatural strain put upon them by the excessive feeding of diabetic patients, without at length resenting the ill-usage. Epigastric pains and a sense of sinking at the scrobiculus cordis, flatulence, and occasional vomiting, are the most common symptoms. As a rule, the bowels are constipated, and require artificial aid to promote their action. The feces are generally pale. In advanced cases diarrhœa not unfrequently occurs, sometimes of a dysenteric character. This is a formidable and generally speedily fatal complication.

The *mental state* suffers a marked alteration in confirmed cases; but the degree and type of it vary a good deal. The change most commonly observed is a heaviness and apathy, a disinclination to mental and bodily exertion, sometimes a positive drowsiness. The natural firmness of the character gives place to a deplorable pusillanimity and a want of moral sense, which are foreign to the individual in a state of health. Persons who previously have been above every equivocation or concealment, resort to petty cunning and positive untruthfulness to deceive their medical attendant as to their food and drink. Nevertheless, in most cases the intelligence itself is not troubled, and continues clear to the end.

The *sexual functions* undergo a notable declension of vigor in advanced cases; and there is frequently actual impotence from failure of the power of erection. This defect, however, is not a permanent one; and if amelioration take place the virile powers return early. Exceptions to this rule also occur. Dr. Prout mentions an instance of a confirmed diabetic, who married and had two children, though the saccharine condition of the urine still persisted.

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[1] Magitot has recently pointed out that an alveolar periostitis is very common in diabetes, and leads to the loss of the teeth, and in severe cases to gangrene of the gum. (Bull. de l'Acad. de Méd., 2d series, vol. x. No. 52.)

COURSE, DURATION, TERMINATION.

Diabetes is an essentially chronic disease; its course is measured by months and years. The ordinary duration of diabetes is from one to three years. Sometimes the disease runs a rapid course and terminates in a few months or weeks. The most rapid example which I have seen was a shopkeeper aged thirty-four. He was perfectly healthy until June 9, 1875. He was then suddenly seized with intense thirst, and profuse discharge of highly saccharine urine, and died on June 18, after an illness of only eight days. I saw another case, a child of three years, who died in three weeks. Becquerel mentions the case of a boy of nine years who died in six days. On the other hand, cases sometimes run on for six, eight, or ten years. The following table shows the duration of diabetes in 100 fatal cases, collected by Griesinger:

Under 3 months	1 case.
Between 3 and 6 months	2 cases.
" 6 " 12 "	13 "
" 1 " 2 years	39 "
" 2 " 3 "	20 "
" 3 " 4 "	7 "
" 4 " 5 "	2 "
" 5 " 6 "	1 "
" 6 " 7 "	2 "
" 7 " 8 "	1 "
Undetermined	12 "

The progress of diabetes is usually equable and continuous; but cases are met with, not very unfrequently, in which the symptoms intermit—the saccharine state of the urine ceasing and recurring at intervals. Dr. Bence Jones[1] has published an account of several such cases in old persons; and I have encountered similar ones in my own practice. These will be again noticed among the milder types of the disease. Girard records an example of intermittent diabetes in a girl of eighteen.[2]

The symptoms sometimes begin suddenly, and not insidiously. Not unfrequently too, the symptoms are much more violent in the first few months than at a later period, when the disease has become confirmed. When diabetes has already existed two or three years the thirst and voracity rarely maintain their primitive intensity. This change from a more acute to a more chronic state must not, of course, be mistaken for a real improvement.

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[1] Med.-Chir. Trans., vol. xxxvi.

[2] Union Méd., 1855

Diabetes may occasionally terminate in complete cure, but the majority of cases end fatally. The disease only exceptionally proves fatal through its own intensity. More frequently one of the complications supervenes and carries off the patient.

COMPLICATIONS.

The most common and formidable complication is pulmonary tubercle, which affects nearly one-half of the cases protracted to the third year. The pulmonary disease runs the course of rapid phthisis. A low and fatal type of inflammation is also liable to arise in the lungs, pleura, or peritoneum. In every tissue of the body there exists a tendency to asthenic inflammation, apt to run on to abscess, diffuse suppuration, sloughing, phagedænic ulceration, or gangrene.

The occurrence of boils and carbuncles in diabetic patients has long been known. The statement of Prout that sugar is always present in the urine of patients suffering from boils, is certainly incorrect. Dr. Goolden mentions the case of a medical man, long diabetic, who had an enormous anthrax on the nucha, which compromised his life. He recovered, however, from the anthrax, and with its disappearance the sugar departed from the urine. P. Frank cites an almost similar case. Phillipeaux and Vulpian ("Gaz. Hebd.," Dec. 6, 1861) relate an example of anthrax in a hemiplegic patient who was not previously diabetic. During the suppuration of the anthrax the urine became strongly saccharine; but ceased to be so when the anthrax cicatrized.

Gangrene of the lower extremities, resembling gangræna senilis, has been several times observed in diabetes. Two examples have come under my observation of fatal gangrene of the great toe in diabetic subjects. One was a man of seventy, who had been diabetic for twelve years. No pulsation could be felt in the arteries of the corresponding limb below the popliteal. The other was sixty years of age, and was not suspected to be diabetic until the toe became gangrenous. Sir H. Marsh[1] mentions the case of a lady, about seventy years of age, suffering from diabetes of long standing, who was carried off suddenly with gangrene of the foot and leg. On examination an obstruction was found in the iliac artery. Dr. Colles (quoted by Marsh) had seen two similar cases of obstructed arteries, and fatal gangrene in diabetes. In 1845, Carmichael presented to the Pathological Society of Dublin two cases of senile gangrene of the lower limbs in diabetic patients.[2] Marchal de Calvi has drawn attention to the same subject more recently, and published four new cases. Additional cases have also been brought forward by Hodgkin, Landouzy, Champouillon, Billard,[1] and others.

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[1] Dublin Quarterly, 1854.

[2] Med. Gaz., 1846, p. 110.

Defects of sight in connection with diabetes have of late years attracted a good deal of attention. They consist either in an enfeeblement of vision (amblyopia), or cataract.

Amblyopia occurs, according to Bouchardat, in about a fifth of the cases of diabetes. Generally speaking, it is slight in degree and temporary, or recurrent. In one of Griesinger's patients the amblyopia ceased when a flesh diet was used, but it was succeeded, shortly after, by cataract. Permanent amblyopia is less frequent; it occurs only in advanced cases, and is a sign of approaching fatal termination. The dimness of sight steadily increases, and at length ends in total blindness.

Occasionally the amblyopia may be accompanied by no ophthalmoscopic signs. In some cases simple atrophy of the optic nerve is seen, while in others there are observed hemorrhages in the retina with consequent retinitis. Opacities of the vitreous are very common.[2]

Diabetic *cataract* was first noticed in this country by Mr. France, and Lécorché has given an excellent résumé of what was known on the subject up to 1861. It occurs generally in inveterate cases of long standing. It is an unfavorable sign; and death follows its appearance usually in a few months; but sometimes patients with diabetic cataract survive for years. The frequency of cataract in diabetes has been estimated very differently by different authors. Griesinger observed cataract in three out of his own seven cases. V. Graefe estimates the proportion as one in five; Bouchardat as one in thirty-eight. Garrod had not encountered cataract in any of the large number of cases of diabetes which he had seen. Out of forty-five cases (written in 1865) which I have treated, only one had cataract. Of 225 cases collected by Griesinger, cataract occurred in twenty.

Diabetic cataract comes on, sometimes without previous defect of vision, sometimes after one or more attacks of temporary amblyopia; sometimes it complicates permanent amblyopia. It generally arises after the diabetic state has lasted eighteen months or two years; but it has been known to appear in six months. Its appearance may coincide with aggravation, amelioration, or stationary condition of the proper diabetic symptoms. Its course is rapid; the two eyes may become completely cataractous in a few days; sometimes it is developed more slowly. It begins in one eye—generally the right—but soon involves both. In the case which I observed, a woman, twenty-four years of age, had been diabetic for two years and a half. Three months previously the left eye became suddenly cataractous: in less than a week the opacity had reached its maximum. The right eye was still clear and vision perfect.

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[1] *See* Charcot, Gaz. Hebd., Aug. 1861; and Dr. Hodgkin, Assoc. Med. Journ., 1854. The whole subject is treated exhaustively by Marchal de Calvi in Recherches sur les Accidents Diabétiques, Paris, 1864.

[2] For further information on this subject, reference may be made to Dr. Gower's "Medical Ophthalmoscopy."

Cataract in diabetes is nearly always of the soft kind; but examples of hard diabetic cataracts have been met with by V. Graefe, Guèrsant, and Sir W. Wilde.

It has been conceived by Weir Mitchell[1] and Dr. Richardson[2] that diabetic cataract is produced by physical imbibition into the lens of the saccharine matter of the aqueous humor of the eye. This opinion is based on the temporary opacity produced in the crystalline lens of the frog, when the animal is immersed in a saccharine solution, or when a similar solution is injected into the cellular tissue. It is very doubtful, however, whether the two conditions are really pathologically analogous. Lécorché failed to produce opacity in the lens of rabbits by injecting syrup into the eye. Artificial cataract in the frog speedily disappears when the animal is removed from the saccharine solution: but diabetic cataract is permanent, and does not disappear when amelioration of the symptoms takes place. How, on the imbibition theory, can the cases be explained, in which (as in the instance which occurred to myself) one lens has been completely opaque for months while its fellow still remains perfectly transparent? How also should its occurrence be (as a rule) so long delayed, and arise so suddenly, without any corresponding increase in the quantity of sugar in the urine? It seems more probable that diabetic cataract is one of the many degenerations of a low inflammatory type so common in confirmed diabetes.

Hepp failed to find sugar in a cataractous lens removed from a diabetic patient. Fisher obtained a similarly negative result in another case. But Stœber found sugar in a lens examined by him.[3]

Operations for diabetic cataract generally fail, from uncontrollable suppuration of the eyeball. Sometimes, however, the operation succeeds; and if the primary complaint be stationary and quite uncomplicated, operation may be recommended as a possible solace to the remainder of life.

Lécorché has described endocarditis as a complication of diabetes. It occurs in the later stages of the disease, and usually affects the auricular surface of the mitral valve. It is ascribed to the irritation of the endocardium by the sugar contained in the blood.[1]

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[1] American Journ. of Med. Sci., Jan. 1860.

[2] Brown-Séquard's Journ. de Physiologie, July, 1860.

[3] Annales d' Oculistiques, xlviii. p. 192.

Very various affections of the *nervous system* may arise as complications in diabetes. Neuralgias are very common; they most frequently affect the fifth cranial and the sciatic nerves, and are in many cases bilateral.[2] Anæsthesia and various forms of paræsthesia have been observed. The affections of the eye have already been described. Deafness, too, is occasionally met with. Amongst the affections of the motor system, we find cramps, in a few cases convulsions, and paralysis of different parts. The paralysis may appear as hemiplegia, or may attack single groups of muscles, such as those of the eye—then producing ptosis or strabismus.[3] The general mental state in diabetes has been already mentioned (*see* p. 252). Further psychical disturbances are sometimes met with. Legrand du Saulle has found in various cases, melancholy, suicidal tendency, apathy, and occasionally imbecility.[4] Dr. Pavy also, has reported a case of temporary mania occurring in the course of diabetes ("Med. Times and Gazette," 1879, ii. p. 262).

The *Coma* which attacks diabetic patients has in recent years attracted considerable attention from clinical and pathological observers. Although this complication had been noted previously by many, yet a complete description was first given by Kussmaul[5] in 1874. Since that time numerous cases have been recorded, and it is now possible to classify them into various groups according to the different clinical pictures presented. Dr. Dreschfeld, in a paper read before the Manchester Medical Society in 1881, analyzed more than 50 cases, and showed that they might be divided into three groups.[6]

In the cases of the first group, the patient after complaining for a short time of headache, or of pain in the epigastrium, with a slight nausea, is attacked by a great feeling of anxiety or by restlessness and some delirium, after which a peculiar dyspnœa sets in. The dyspnœa affects both the inspiratory and expiratory stages of respiration, but no cause can be found for it, by examination of the chest, and there is usually no cyanosis. This form of dyspnœa has been aptly termed by Kussmaul "Air-hunger." Comatose symptoms appear, from which the patient may at first be roused by shouting in his ear, but gradually the coma becomes complete. The pulse is much quickened and the temperature is lower than normal. Convulsions are of exceptional occurrence. The state of the pupils varies, but commonly they are dilated. The coma generally ends in death within 48 hours, and only a few cases have been known to recover.

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[1] *See* Brit. Med. Journ , 1882, i. p. 843. (I have met with one example of this form of endocarditis in the post-mortem room of the Manchester Royal Infirmary. It corresponded entirely with the description given above. R. M.)

[2] Worms. *See* Lancet, 1878, ii. p. 708.

[3] *See* Ross, Diseases of the Nervous System, vol. ii. p. 933.

[4] Gazette des Hôpitaux, 1877

[5] Deutsches Arch. f. klin. Medicin, Bd. xvi. S. 1.

[6] *See* Brit. Med. Journ., 1881, ii p. 710. A similar classification is also adopted by Frerichs in his recent treatise on Diabetes.

The symptoms of the second group of cases closely resemble those of uræmia from kidney disease. The dyspnœa is not so well marked as in the preceding form. The patient complains of general weakness, becomes drowsy, lapses into a state of coma with low temperature and small pulse; and soon dies.

The third form shows all the symptoms of acute Alcoholic Intoxication. The patient does not show the same muscular weakness as is observed in the two preceding forms. He is attacked suddenly by great excitement, staggering gait, and drunken delirium. Gradually drowsiness and coma develop as in the other forms and death ensues.

Certain phenomena are common to all three forms. During and preceding the attack, a peculiar sweet smell, likened to that of chloroform, is perceived in the breath. The urine has a similar odor; and it gives the so-called "acetone reaction" with perchloride of iron—that is, when a solution of perchloride of iron is added, in excess, a deep red color is produced, almost like that of port-wine.[1] This reaction was first described by Gerhardt. The urine, during the attack, generally contains less sugar than before the development of the symptoms, and not uncommonly shows a little albumen.

Coma is more liable to occur in the earlier stages of diabetes and in young subjects. It is especially frequent after muscular exertion and after the first adoption of a diabetic diet. Constipation, which is common in all diabetics, seems to predispose to the development of coma.

The pathology of diabetic coma is still in the region of theory. The various views which are held to explain the development of the symptoms may be shortly summarized as follows:

Kussmaul formulated the view that it was due to the presence of acetone in the blood. The same substance being, as he believed, the cause of the peculiar odor of the breath, and of the characteristic reaction of the urine with perchloride of iron. He experimented by giving acetone to animals, but only after very large doses did he get any symptoms, and even then, although he believed that the symptoms were those of diabetic coma, they have since been shown to differ from it in essential particulars. Large doses of acetone given to man produced no effects. Acetone has in some cases of diabetic coma been absent from the blood, while the acetone odor of the breath is found in many other conditions, especially in digestive disturbances, in tuberculosis, and in many disorders of children, when no coma occurs.

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[1] Von Jaksch has shown that the so-called "acetone reaction" is by no means confined to diabetic urine. He has found it in various febrile disorders and in several other conditions. (Zeitschr. f. Klin. Med., Bd. v.)

Dr. Balthazar Foster and Dr. Saundby[1] thought it probable that the acetone present in the blood might act by dissolving the red blood-corpuscles. In one case, under the care of Dr. Foster, the blood was noticed to be of a pale lake color, and, on standing, a grayish layer formed on the top. This layer was composed of granular material, and did not dissolve in ether. A similar appearance was produced experimentally by mixing acetone with blood.

Rupstein[2] showed that free acetone did not exist in the blood, but that it was produced by the breaking up of ethyl-diacetic acid into acetone and alcohol. It is quite possible that cases of the third group may actually be due to what they resemble—acute intoxication with alcohol.

Quincke[3] pointed out, that the substance contained in the urine, resembles acetic ether, in its reaction with ferric chloride, and in breaking up into acetone and alcohol, but that the urine does not smell of acetic ether, and does not yield acetic ether on shaking with ether. He showed also that if acetic ether is present, it is combined with something which is neither grape-sugar, an alkali, nor a normal constituent of the urine. Rupstein and Salomon were unable to obtain the ferric chloride reaction from the condensed breath. Acetic ether was also given to dogs, without producing any poisonous effects. Still another substance will give the ferric chloride reaction in urine, and this is diacetic acid or aceto-acetic acid. Deichmüller and Tollens[4] have asserted that this is the substance present in the urine of diabetics. Brieger, however, was again unable to produce poisonous symptoms in dogs by giving this substance.

Quite recently Stadelmann[5] has found an acid in diabetic urine which is probably β croton acid, and has suggested that diabetic coma might be due to poisoning by this. Against this view is, however, the fact that Frerichs (loc. cit.) has found the alkalescence of the blood undiminished in diabetes. β croton acid was also found to be practically non-poisonous.

The blood of diabetics has been often observed to contain excessive quantities of fat (*see* p. 263). Professors Sanders and Hamilton[6] described in 1879 a case of diabetic coma, in which large quantities of fat were found in the blood and numerous fat emboli were seen in the lungs. They suggested that fat emboli in the lungs and brain were the cause of the symptoms. Similar cases have been described by Starr,[7] Fitz,[8] and by Fraser and Logan.[9] Dr. Dreschfeld, however, was unable to find fat emboli in four cases of diabetic coma, although the blood contained large quantities of fat. Dr. Taylor[1] obtained a similar result in three cases. Moreover, fat emboli are found in other disorders without producing any such alarming symptoms.

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[1] Brit. Med. Journ., 1878, vol. i. p. 78.

[2] Centralbl. f. Med. Wissen., 1874, p. 865.

[3] Berlin. klin. Wochensch., 1880, p. 1.

[4] Annalen der Chemie, Bd. ccix. p. 22. Also, Brit. Med. Journ., 1882, i. p. 665.

[5] Archiv f. exp. Patholog., Bd. xvii. p. 443.

[6] Edin. Med. Journ., July, 1879, p. 47.

[7] New York Med. Record, vol. xvii. p. 477.

[8] Boston Med. and Surg. Journ., vol. civ. p. 124.

[9] Edin. Med. Journ., Sept. 1882.

Schmitz,[2] of Neuenahr, propounded the view that the fatty heart, so often found in diabetics, was the cause of the comatose symptoms. Teschemacher[3] believes that a lesion of the sympathetic may be present, which might cause shock. Various other views have been propounded, such as that the symptoms are due to thickening of the blood from the presence of excessive amounts of sugar, to uræmia, to cerebral anæmia, etc., but none of these has received any support.

Ebstein has described a necrosis of epithelium as occurring in diabetes in various glands, and amongst others in the kidney.[4] There it is seen that the epithelium of the convoluted tubes and of Henle's loops was swollen and hyaline in character. In many parts the cells are fatty, the nuclei are indistinct and do not easily take up coloring matters, and the cells are sometimes detached from the wall of the tubule.[5] It seems most probable that this kidney lesion may cause a retention in the blood of various poisonous matters, which are derivatives of sugar, but whose nature is as yet unknown.

PATHOLOGY AND MORBID ANATOMY.

Although diabetes is a frequently fatal disorder, necrology has thrown but an uncertain light on its seat and nature.

The more palpable anatomical changes which have been often found in the lungs and elsewhere, are manifestly not due to the disease itself, but to some of its numerous complications. Physiological data would lead us to look for the primary seat of diabetes in the liver itself, or in some part of the brain or nervous system connected with the liver.

The liver has certainly not yet given up its secret, if it have any. The accounts of its appearance, after death from diabetes, are contradictory. Its size is usually normal; sometimes it is a little enlarged, sometimes a little atrophied. In some cases it is congested, in others the reverse. Occasionally, it contains a good deal of sugar—more frequently, none at all. Dr. Wilks believes that the diabetic liver presents differences to the eye which enable it to be distinguished from others: it is firm, tough, homogeneous or uniform in appearance, and dark in color.[6] But other descriptions are quite at variance with this. Griesinger found the liver granular and easily torn in one case;

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[1] Guy's Hosp. Reports, 3d series, vol. xxv. p. 158.

[2] Berl. klin. Wochensch., 1876, p. 63.

[3] Berlin. klin. Wochensch., 1881, p. 450.

[4] Deutsches Arch. f. klin. Medic., Bd. xxviii. p. 143.

[5] (I have found this lesion in three consecutive cases of diabetic coma, which died in the Manchester Infirmary. R. M.)

[6] Pavy on Diabetes, p. 140.

in fourteen others the livers were perfectly normal. Dr. Dickinson sees in the livers of diabetic patients evidence of long-continued hyperæmia, ending in overgrowth of the epithelium and fibrous tissue.

Microscopical investigations have been equally unsatisfactory. Förster and Griesinger found the liver-cells natural. Beale[1] and Frerichs[2] remarked a diminution or absence of fat. Pavy found the fat undiminished. Stockvis found the fat undiminished, but an unusual proliferation of the hepatic cells. Ehrlich[3] has found excess of glycogen in the interior of the liver cells.

Of the bile, Dr. Pavy remarks, that in nearly all the cases in which he has specially examined it, it has presented a striking appearance, resembling a rhubarb mixture, and depositing a copious sediment consisting of columnar epithelium, and yellow, amorphous, granular-looking matter.

Prout says that he frequently observed a gorged condition of the veins terminating in the portal system.

Affections of the pancreas have been frequently found in diabetes, and especially hardening and atrophy of the organ. The latter condition has been especially insisted upon by Lancéreaux.[4]

The researches of Bernard on artificial glycosuria, and the large number of recorded cases of diabetes in which the disease has followed or coincided with disease or injuries of the brain, have directed the attention of pathologists to a more minute examination of the nervous centres.

The floor of the fourth ventricle has been examined in several instances. Sometimes it has been found quite natural; in other cases it was found the seat of serious pathological changes. In 1860, Luys brought before the Société de Biologie an example in which this spot was softened, highly vascular, and of an unnatural brown color. The nerve-cells were found degenerated and full of yellowish granules.[5] Monneret followed up this observation with another in which similar changes were encountered in an earlier stage.[6] Tardieu records a case of diabetes in which there existed slight paralysis of the left side for three months: the diabetes persisted until death two years afterwards from phthisis: the medulla oblongata was found congested and of a dark gray color.[7]

In his work on diabetes, Dr. Dickinson describes the changes in the nervous system found by him in eleven fatal cases. A practised and careful eye, he says, will often detect a porosity in limited patches of the white matter, as if it was closely beset with pin-holes, each puncture containing a vessel much smaller than itself. Under the microscope these excavations become conspicuous objects. The excavations are found about arteries or in positions which arteries have once occupied. They are sometimes globular, and at their maximum such as would lodge peas. Often they are elongated and narrow, and may be described as tunnels directed by the course of the vessels. They are caused by a destruction and absorption of the nervous matter along the course of the arteries, and are, at least in some instances, caused by an escape of the contents of the vessel into the surrounding tissues, with consequent degeneration, softening, and removal of the nervous matter which has been permeated by the intrusion.

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[1] Med.-Chir. Rev., 1853, p. 226.

[2] Cited by Griesinger, loc. cit. p. 34.

[3] *See* Frerichs on Diabetes, 1884.

[4] Bull. de l'Acad. de Méd., 1877.

[5] Bulletin de la Soc. de Biol., 1860.

[6] Gaz. des Hôp., Jan. 11, 1862.

[7] Med. Times and Gaz., Feb. 1862. *See* also above, p. 259.

When the disease has proceeded to its natural end, the excavations are widely scattered through the brain; numerous, small, and closely set in the white matter of the convolutions, fewer and larger about the central parts. The corpora striata, optic thalami, pons, medulla and cerebellum are the chosen seats for the largest and most striking holes. The white matter of the convolutions is often rendered strikingly cribriform, in patches, by numerous erosions. More than fifty have been found in a space of not half an inch square—minute, but distinctly visible to the naked eye. Each contains a vessel, usually an artery, around which is an irregular interval, containing crystals of hæmatine and products of nervous degeneration. These holes are evidently exaggerations of the perivascular spaces. The cavities, whatever their size and place, are much the same in appearance and contents. They are abruptly bounded, usually with a narrow margin of disintegrated nervous tissue, the brain matter outside of this being absolutely natural. According to their date, they contain degenerate remains of nerve tissue, remnants of bloodvessels, or of extruded blood, or are empty. The products of nervous degeneration are first removed, then for a while the cavity contains only dilated or shrunken and obsolete arteries, with areolar tissue derived apparently from the perivascular sheath, and crystals of hæmatine. Lastly these disappear also, and a mere vacuity is left. The changes in the cord are similar to those in the brain, but less declared.

Dr. Dickinson regards these lesions as the initial pathological fact of ordinary idiopathic diabetes, and as the primary cause of glycosuria.[1] Whether these observations, and the deductions drawn from them, will stand the proof of controlling inquiries, remains to be seen. They have not, even now, been allowed to pass without challenge. Külz[1] states that in three cases of fatal diabetes examined by him no enlargement of the perivascular canals of the medulla could be discovered, neither with the naked eye nor the microscope. The same author cites W. Müller as having carefully compared the medulla in three diabetics and in two cases of paralytic dementia, and as coming to the conclusion that diabetes may exist without enlargement of the perivascular canals, and that enlargement of the perivascular canals may exist without diabetes, and, consequently, that the anatomical changes found by Dickinson have no causal connection with diabetes. Taylor and Goodhart[2] have also been unable to find these appearances.

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[1] Dr. Dickinson seems to have given up the claim that these changes are peculiar to diabetes. *See* Lancet, 1878, i. p. 117.

Dr. Dreschfeld made a communication on this subject to a meeting of the Manchester Medical Society, on Dec. 1, 1875. He had examined the brains of two fatal cases of diabetes. In both these cases the brain and medulla were most carefully searched, and microscopical sections were made both from fresh and hardened preparations, but in none of them could alterations in the perivascular canals be discovered, such as have been described by Dr. Dickinson. Indeed one of the specimens had been used by Prof. Gamgee to illustrate before his class the normal histology of the brain. It certainly seems strange, if this wide-spread destruction of nervous matter really occurs in diabetes, that mental aberration and paralytic accidents should usually be so conspicuously absent from the clinical history of idiopathic diabetes.

Silver and Irvine[3] have described hemorrhage and softening of the spinal cord in the cervical and upper dorsal regions, in one case.

Lesions of the pneumogastric and of the sympathetic nerves have also been found in some cases of diabetes. Harley cites a case in which a calcareous tumor as large as a hazel-nut was found pressing on the right vagus in the chest. Poniklo[4] has described an overgrowth of fibrous tissue in the ganglia of the sympathetic.

The *blood*, as might have been expected, is unnaturally charged with sugar in diabetes. It has been found in the proportion of from 0.3 to 0.5 per cent. From the blood, sugar finds its way into all the tissues and fluids of the body. Sugar has been found in the feces, the sweat, the humors of the eye, the tears, the saliva, and the gastric juice.

In many cases of diabetes the blood also contains a large excess of fat. Dr. Gamgee, in one case, found as much as 10.8

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[1] Beiträge z. Path. u. Therap. d. Diab. Mell. Marburg, 1874, p. 10.

[2] Guy's Hospital Reports, vol. xxii. p. 415.

[3] Path. Trans., vol. xxix.

[4] Lancet, 1878, i.

per 1000—the normal proportion being about 2 per 1000. (Gamgee's "Physiological Chemistry," vol. i. p. 172.)

Of the secondary lesions or complications, those found in the lungs are the most common. Out of sixty-four autopsies collated by Griesinger, tubercles were found in the lungs in thirty-one, or nearly one-half. Pavy and Wilks believe that the pulmonary mischief is not always genuine phthisis, even when it runs a closely similar course, but consists in a chronic inflammation leading to the breaking down of the lung tissue and the formation of cavities.

Pneumonic consolidation and gangrene of the lungs have likewise been not unfrequently found. Sometimes (not always) gangrene of the lungs in diabetes is not accompanied by the characteristic fetor of that complaint.

In long-standing cases, the kidneys are not unfrequently found seriously altered. Out of Griesinger's sixty-four autopsies renal alterations were found in thirty-two. In seventeen instances there was degeneration resembling some form of Bright's disease, mostly with fatty degeneration of the renal epithelium. Granular atrophy of the kidneys was never found; but cysts, cicatrical spots, adhesions of the tunica propria, and pyelitis. In five cases the kidneys were markedly hyperæmic, and in seven considerably hypertrophied. Prout states that the kidneys of persons dying of diabetes assume frequently a peculiar deep orange tint on exposure to the air. Ehrlich has recently described collections of glycogen in the cells of the kidney especially in the region where the cortical and medullary portions join.[1]

The stomach is commonly found distended, and the mucous membrane thickened and softened.

PHYSIOLOGICAL AND THEORETICAL CONSIDERATIONS RELATING TO DIABETES.

Much light has been thrown on the pathology of diabetes by the observations of Bernard and others on the presence of an amyloid substance in the liver, and the possibility of inducing glycosuria in animals by artificial means. A résumé of the present state of these questions is absolutely necessary to the comprehension of any theoretical view of diabetes.

Bernard discovered the fundamental fact, that the liver of all healthy animals contains a large quantity of a substance resembling starch or dextrine. When the liver of an animal newly killed is abandoned to itself in a warm place, it speedily becomes charged with sugar by the conversion of a portion of this substance into glucose; and when the sugar so produced is washed completely out by a stream of water, the organ abandoned to itself as before, becomes, again in twenty-four hours, abundantly charged with sugar. This conversion goes on until all the amyloid substance is changed into sugar. The transformation here witnessed takes place by the action of a peculiar ferment which circulates in the blood.

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[1] See Frerichs, loc, cit.

I have obtained the amyloid substance of the liver (which has received the various names of *animal* or *hepatic dextrine* (or *starch*), *hepatine*, and *zooamylum*) in the greatest purity and with the greatest ease from the liver of the oyster. The large fawn-colored mass, which constitutes the delicacy of this mollusk, should be cut out, and plunged for a few minutes into boiling water. The hardened mass is then pounded in a mortar, mixed with a small quantity of water, and boiled so as to form a decoction. This is subsequently filtered, and poured into five times its bulk of strong alcohol or glacial acetic acid. An abundant precipitation of snow-white flakes is produced. This is the amyloid substance. It is collected on a filter, washed with alcohol and dried.

When pure, hepatic dextrine is a white, tasteless, inodorous body. It dissolves freely in water, forming an opalescent solution like skimmed milk. It contains no nitrogen; its formula is $C_{10}H_{10}O_{10}$. With iodine it behaves like vegetable dextrine, yielding a deep wine-red coloration. It does not reduce the salts of copper, nor ferment with yeast; but (like starch and dextrine) it is speedily converted into glucose by the contact of saliva, pancreatic juice, or diastase. It is similarly converted by the contact of fresh blood, which has no effect on starch and dextrine. This last property is its peculiar characteristic.

Schiff has satisfied himself, by a most ingenious set of experiments, that he has detected the exact situation and physical condition of the amyloid substance in the liver. He found it, not infiltrated or dissolved in the hepatic tissue, but collected into separate vesicles or granules precisely as occurs with starch in the vegetable kingdom. In the frog, he found, under a magnifying power of 600 diameters, that the liver-cells, in addition to one or two round nuclei and twelve or twenty fat globules, contained an immense number of minute pale vesicles varying in size from $\frac{1}{1600}$ to $\frac{1}{1000}$ of a line. Within these vesicles the amyloid substance is accumulated. The outer membrane (as in the vegetable starch-granule) contains nitrogen. Nasse[1] has subsequently detected similar amyloid vesicles in the liver-cells of worm-blooded animals. They differ from most of their vegetable homologues in not possessing concentric markings, and in not yielding a blue coloration with iodine. I have endeavored to verify these observations in the liver of the frog and the oyster, but without success.

Animal dextrine is always present in the livers of all the healthy animals hitherto examined, whether living on vegetable or animal food, or fasting. But under a variety of diseased and unnatural conditions it quickly disappears. The circumstances preceding death from disease, are such that the liver scarcely ever contains a trace of amyloid substance when examined *post-mortem*.

The *peculiar ferment* of the liver-dextrine exists in the blood, but has not yet been isolated. It is not liable to disappear under those conditions of disease which cause the hepatic dextrine to vanish so quickly. Nevertheless, it is sometimes absent. Schiff made the curious discovery that this ferment totally disappears from frogs during the second half of the winter and the early spring months. This occurs as a regular event in the annual changes which these batrachians undergo. During this interval the liver is as full as usual of amyloid substance, but no spontaneous production of sugar occurs when the organ is abandoned to itself in a warm place, and artificial glycosuria cannot be engendered in such animals. When, however, the blood of another animal, which is not in this peculiar condition, is injected into the bodies of winter frogs, or applied to their livers, the usual production of sugar takes place rapidly. This absence of a ferment has not been noted, as a regular occurrence, in any warm-blooded animals; but Dr. Pavy states that he once encountered a healthy rabbit in this condition.

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[1] Archiv des Vereins fur gemeinschaftl. Arbeiten, IV. I., p. 97.

Great divergence of opinion prevails as to the destiny of the liver-dextrine during healthy life. Many physiologists adhere to the view of Bernard, and believe that a continual conversion of this substance into sugar is going on in the liver, and that a quantity of sugar is being constantly poured into the hepatic veins and carried off into the circulation.[1] Dr. Pavy, on the other hand, contends that there is no conversion of hepatic dextrine into sugar during healthy life, nor any continual stream of sugar flowing into the circulation; and that when such conversion does take place, it is an abnormal or diseased occurrence, or due to *post-mortem* changes.

"Instead of the liver being essentially a sugar-forming, it is a sugar-assimilating organ. Its great function in relation to sugar is to prevent this principle reaching the circulation to any material extent." (Pavy's Croonian Lectures.)

In cold-blooded animals, the view of Pavy has appeared to me to be the correct one. I have repeatedly tested the point in frogs and oysters, and have never succeeded in detecting a trace of sugar in the liver, if the organ was examined before the possibility of any *post-mortem* changes. In the case of the oyster, the experiment is a very easy one. A fresh oyster is cut in half with a pair of scissors, in such a way that one half shall drop into a capsule of boiling water; the other half is laid aside in a warm place. The latter very speedily becomes abundantly saccharine; but in the former the ferment has been rendered inert by the heat, and not a particle of sugar can be detected in it, even after being long kept in a warm place. The conditions in the oyster's liver are precisely similar (as far as is known) to those in the liver of a warm-blooded animal. An abundance of amyloid matter and the ferment coëxist side by side in the organ, and yet no reaction takes place between them, and no sugar is produced, so long as the normal state is maintained.

A similar experiment is more difficult in warm-blooded animals, because it is impossible to proceed with the same celerity; yet in Dr. Pavy's hands the results obtained were fully confirmatory of his doctrine.

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[1] Among these may be mentioned Harley (Proc. Roy. Soc., vol. x. p. 289); Schmidt, of Dorpat (Comptes Rendus, t. xlix. p. 63), and Lusk (New York Med. Journ., 1870). Dalton, Treatise on Human Physiology, 5th ed. p. 193.

Bernard's glycogenic theory rests chiefly on the fact that in newly killed animals the blood of the hepatic veins has been found sensibly richer in sugar than that of the body generally. Pavy attributes this result to rapid changes which take place during the performance of the experiment. He has varied the proceeding in such a manner as to avoid these disturbances. He catheterized the right heart by introducing a tube along the jugular vein. In this way, if the animal remained quiescent, the blood of the hepatic veins was obtained in its normal state. Hepatic blood so obtained, was found to contain only those minute traces of sugar which exist in every part of the circulation.

Dr. Robert McDonnell,[1] in an admirable series of researches, has repeated and varied the experiments of Pavy, and obtained results which do not seem to admit a possibility of doubt that amyloid substance is *not* converted into sugar in the liver during healthy life. In his memoir on the functions of the liver,[2] McDonnell brings forward some facts and considerations of great weight in support of his view, that the real destiny of the liver-dextrine is to unite with nitrogen (set free by the disassimilation of fibrine and a portion of the albumen of the portal blood) so as to constitute a new protein compound resembling casein, which is being constantly poured into the circulation through the hepatic veins.[3]

Dr. Pavy appears to insist too absolutely on the absence of any unimpeachable evidence of the disappearance of sugar introduced into the blood, except by its removal through the kidneys. It has been fully made out that sugar and dextrine may be injected continuously into the blood in certain small quantities—that is, so much that the percentage of them in the blood shall never rise beyond 0.2 or 0.3—without producing saccharine urine.[4] What becomes of sugar so introduced is doubtful. It may not be oxidized, as has usually been believed, into carbonic acid and water; perhaps it is transformed into amyloid substance and lodged in the liver. That it disappears somehow without escaping with the urine cannot admit of doubt. Some experiments of Schiff appear to bear decisively on this point. He induced artificial diabetes in frogs by puncturing the spinal cord; he then ligatured portions of the liver, so that the discharge of sugar into the circulation was diminished in proportion to the size of the piece of liver included in the ligature. When a piece equal to about a fifth part of the organ was included in the ligature, sugar was still poured into the circulation, *but not in sufficient quantity to produce glycosuria.*[5]

ARTIFICIAL GLYCOSURIA AND DIABETES.—We are led to believe, then, on the evidence above adduced, that although the amyloid matter and its ferment must be in close proximity in the hepatic tissue, they do not come into actual contact and react upon each other during healthy life; but they may be brought into conjunction under a variety of unnatural conditions induced by disease or injury; and physiologists are able to bring about these abnormal conditions at will, and to cause sugar to appear in the urine.

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[1] *See* Proceedings of the Royal Irish Academy, Feb. 13, 1860.

[2] Observations on the Functions of the Liver, by R. McDonnell, M.D. Dublin and London, 1865.

[3] Pavy's results have been further confirmed by Meissner and Jaeger, and by Schiff and Herzen. The whole of this question is treated at length in the 2d ed. of Dr. Pavy's work on Diabetes.

[4] Schiff, loc. sit. p. 134.

[5] That a trace of sugar exists in normal blood (even in flesh-fed animals) seems to be now generally admitted. Bernard, who originally taught this was consumed in the lungs, afterwards believed with other physiologists, that it is utilized in the nutrition of the tissues and especially of the muscles.

Artificial glycosuria may be produced in various ways, namely, by cutting or puncturing diverse parts of the nervous centres and certain organic nerves; by impeding respiration; putting animals under the influence of anæsthetics and tetanizing substances; injecting acid substances into the portal veins; and thrusting needles into the liver.

Most, if not all, of these injuries, different as they appear, act finally in the same manner, and cause dilatation of the hepatic bloodvessels, and consequent hyperæmia of the organ. This dilatation may (conceivably) be brought about in two ways: either by an increased action of the longitudinal muscular fibres (dilating muscles) of the small vessels[1] —this would be an active congestion—or by a paralysis of the circular fibres, whereby the vessels would give way and expand before the propulsive action of the heart.

The contractile tissue of the hepatic vessels, like that of the vascular system generally, is under the control of a distinct nerve-arrangement, with a local centre in its neighborhood (probably the cæliac ganglion), and upward prolongations by the sympathetic and the spinal cord into the cerebral centres. The separate threads of this communication are, in the lower parts of their course, placed widely apart; but they approach in the spinal cord; and in the floor of the fourth ventricle they are collected into a close bundle before their final dispersion into the cerebral hemispheres.

An irritation applied to any part of this nervous communication may cause temporary glycosuria; and in the floor of the fourth ventricle even the puncture of a needle, if it be made exactly at the right spot (midway between the origins of the auditory nerves), is sufficient. The difficulty of exactly hitting this spot renders the operation somewhat uncertain, except on condition of injuring the surrounding parts extensively; and Schiff found it preferable to pass in a needle and destroy the whole thickness of the cord at the point of origin of the brachial nerves. This operation never fails to produce *temporary* glycosuria. In warm-blooded animals, the urine continues saccharine for a few hours; in frogs, about four days. Schiff gives good reasons to believe that glycosuria so produced is caused by an active congestion of the liver.

The *permanent* diabetes, with which practitioners are familiar in the human subject, appears, on the contrary, to be paralytic in its nature, and to be due to a passive congestion of the liver from loss of contractility in the circular fibres of the hepatic vessels. Schiff succeeded in inducing, in rats, a permanent diabetes which may be looked on as the true counterpart of the idiopathic disease in man. This was accomplished by operating on the spinal cord at a lower point. He passed a strong needle into the spinal cord (with the least possible injury to the surrounding parts) and destroyed it, opposite the second dorsal vertebra. Rats operated on in this way lived, provided their temperature was artificially sustained, for seventeen and even twenty days, and continued diabetic to the end. Rabbits sometimes out-lived this operation nine days, and continued diabetic to the last day. Animals higher in the scale than rodents do not survive this operation.

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[1] *See* Schiff, loc. cit. p. 92. *See* also a paper by the same author, Journ. de l'Anat. et de Phys., t. iii. 369. Schiff states that cutting the sciatic nerve, and even the nerves of the anterior extremity, causes a slightly diabetic state in rabbits.

Temporary glycosuria has also been induced by impeding respiration (Pavy); by poisoning with strychnia and woorali; by thrusting needles into the liver (Schiff); by chloroform and ether inhalations in warm-blooded animals; in frogs, by tying the afferent veins of the kidneys so as to increase the flow of blood through the liver (Schiff); by injecting stimulants into the hepatic veins (Harley); by painfully stimulating a sensory nerve, by stimulating the central end of the divided pneumogastric or of the depressor nerve, and by stimulating the first dorsal pair of nerves (Laffont).

Dr. Pavy has found that strongly pronounced glycosuria is produced in dogs by injecting arterial blood into the portal vein. This, he thinks, "affords an explanation of the glycosuria which follows Bernard's puncture of the fourth ventricle and the various lesions of the sympathetic. Without any new agent being called in, sufficient is presented in the state of the blood to account for the production of sugar that occurs. By a vaso-motor paralysis affecting the vessels of the chylo-poietic viscera, the blood will reach the portal system without having become dearterialized in its natural way; and in this state it has been shown to possess the property of acting within the liver in such a manner as to determine the production of glycosuria" ("Proc. Roy. Soc.," June 17, 1875).

It should also be mentioned that the introduction of large quantities of sugar and starch by the digestive organs occasions glycosuria—showing that the assimilating power of the liver over these aliments is not unlimited. Inuline (which replaces starch in the compositæ) induces slight glycosuria, even when partaken of in comparatively small quantity (Schiff).

Bearing these physiological data in mind, we shall not find any difficulty in explaining the circumstances under which temporary glycosuria occurs in the human subject in connection with various injuries and diseases; and we obtain some dim insight into the true pathology of clinical diabetes.

Dr. Pavy applies his views to the explanation of permanent glycosuria in the following manner: He urges that in diabetes there is a hyperæmia of the abdominal organs, and especially of the liver, this being probably caused by some affection of the vaso-motor mechanism. Such a hyperæmia would cause the blood flowing through the liver to be more highly oxygenated than is normal, and would, therefore, produce glycosuria, as in his experimental results.

It must be remembered, in searching for sugar in the urine of persons who present the alleged conditions of glycosuria, that the search will be in vain if there be great disturbance of the general system, and especially if there be fever; for the amyloid substance speedily disappears from the liver under these circumstances, and consequently no sugar can appear in the urine, however perfectly all the other conditions for its occurrence exist. This is doubtless the reason of the many contradictory results of bedside observations on the occurrence of saccharine urine. I have repeatedly examined the urine of patients with obstruction in the chest (emphysema, etc.) in whom there existed great hyperæmia of the liver, without finding sugar; but it nearly always happens in such cases that the general well-being of the patient is deeply affected, or that there is positive pyrexia. From what has been said above, it is important to remember that in such cases, the hyperæmia is of the venous and not of the arterial form.

Although we appear to be approaching an exact knowledge of the pathogenetic elements of glycosuria, it is yet manifestly impossible, in the present state of science, to frame a comprehensive and clear theory of diabetes. It would seem highly probable that diabetes consists proximately in some disturbance of the destiny and function of the amyloid substance of the liver. But this disturbance may be due originally to disease far away from the liver itself, in some part of the nervous circle which controls this function. Occasionally, as in traumatic cases, it is possible to place the finger on the primary lesion; but in the immense majority of cases we are left in a sea of conjecture. Further researches conducted in the light of past and future physiological discoveries, can alone reduce these conjectures to order and certainty.

DIAGNOSIS AND PROGNOSIS

THE DIAGNOSIS of diabetes is generally a very simple matter, when attention is once directed to the urine—the existence of sugar in the urine, and diuresis being the only points to be ascertained. The means of detecting sugar and of estimating its quantity have already been fully considered.

Care must be taken, however, not to conclude too rashly that this formidable disease exists, from the mere finding of sugar in the urine. It has just been shown that the urine becomes temporarily saccharine under certain conditions quite apart from genuine diabetes. Before the existence of diabetes can be deduced, it must be ascertained that there is a considerable quantity, and not a mere trace, of sugar in urine; secondly, and especially, that its appearance is not temporary, but persistent; and thirdly, that there is a less or greater increase in the volume of the urine.

A more recondite diagnosis than this, is at present rarely possible: but it is to be hoped that the time is not very far distant when we shall be able to indicate the seat of the initial lesion in each case, and to refer it to a cephalic, spinal, sympathetic, hepatic, or other category, as the symptoms or previous history may point out.

PROGNOSIS.—The general prognosis is highly unfavorable: the large majority of the cases terminate fatally. A not inconsiderable number, however, recover completely; and many more attain to a state of conditional amelioration—that is, an amelioration which is conditional on the observance of a certain diet and regimen.

The special prognosis depends on a variety of circumstances, of which the following are the more important. The younger the patient the less hope of ultimate recovery. All the cases under twenty, which I have seen, have eventually succumbed. In persons advanced in years, the appearance and persistence of saccharine urine is a far less serious affair: it may continue for many years in oscillating degree with fair preservation of health. It is a curious circumstance that diabetes in corpulent persons is very markedly less formidable than in those of spare habit. Saccharine urine without diuresis is far less serious than when the urine is abundant. *Cæteris paribus*, the longer the disease has existed, the more unfavorable the prognosis; *cæteris paribus*, also, the greater the general severity of the symptoms, the less is the hope of amendment. The ascertained cause of the disease also affects the prognosis. Cases which can be traced to mental anxiety and traumatic lesions appear to be somewhat more hopeful than those for which no tangible cause can be assigned.

The presence of albumen in the urine, of thoracic or intestinal complications, are fatal signs. The existence of permanent amblyopia, or cataract, is a very unfavorable indication. Such cases generally terminate fatally within six or twelve months, and, so far as is now known, always eventually; that is, they are essentially incurable cases, though some of them survive many years.

The results of treatment furnish important data for estimating the gravity of the prognosis. A very favorable circumstance is the disappearance of sugar from the urine when saccharine and starchy matters are withdrawn from the diet. Even great diminution without total disappearance of sugar is a hopeful sign. On the other hand, the persistence of sugar in quantity on a purely animal diet is a sign that the disease is confirmed and far advanced. A moist perspirable skin, a fair appetite, a stationary condition, are all favorable signs.

It must be remembered that when, by treatment, the disease has been brought apparently to a stand-still, a diabetic patient still holds his life by a very frail tenure. To use the expression of Dr. Prout, persons with confirmed diabetes, though apparently in fair health, live as it were on the brink of a precipice. A little undue exposure to wet or cold, an unusual bodily exertion —trifles to the healthy—may excite inflammatory complications which prove rapidly fatal.

TREATMENT.

The seat and nature of the primary lesion are, as we have seen, nearly always concealed; and we know almost nothing of what may be called a radical treatment of diabetes. But the more prominent symptoms—thirst, inordinate appetite, emaciation, and the copious diuresis, are unquestionably dependent, in great part, on the accumulation of sugar in the blood, and the imperious necessity for its removal. A clear indication for treatment, therefore, is to diminish this accumulation. In our ignorance of any direct means of checking the formation of sugar in the body, we resort to the indirect method of withdrawing sugar and amylaceous substances (which are easily converted into sugar in the *primæ viæ*) from the dietary. We endeavor further to combat any existing disorders of the skin, stomach, bowels, and other internal organs, and to allay certain troublesome symptoms which arise in the course of the disease. By means of a regulated diet, clothing, and habits of life, invaluable help can be rendered to diabetic patients: sometimes so as to open the way to perfect recovery: often, nay generally, so as to relieve suffering and prolong life.

DIET.—The plan to be pursued is to withdraw as completely as possible, but not too suddenly, all saccharine and amylaceous articles—the chief of which are bread and potatoes—from the diet, and to replace them by appropriate substitutes from the vegetable kingdom, and by animal food.

It is well known that human life can be sustained in perfect vigor on a purely animal diet. Ths inhabitants of the arctic regions subsist exclusively on the flesh and blubber of seals, fish, and such produce as the chase of the climate affords. The fur-hunters of British America exist for many successive months, leading a life of great muscular activity, on a flesh diet alone. But in our more settled communities the use of bread and potatoes is almost a second nature, and deprivation of them is, to the great majority of individuals, an almost unendurable hardship. To obviate this difficulty several substitutes for bread have been contrived which are of very great value in the management of diabetes.[1]

In the choice of substances from the animal kingdom, the only doubtful or forbidden articles are milk, honey, and liver. Butcher's meat, cheese, butter, fat and oil, poultry, game, eggs, fish, may be used freely in any form. Broths, soups, and jellies, (prepared without meal or sugar) are also permissible *ad libitum*. Milk, which contains considerable proportions of a saccharine substance, should, as far as possible, be replaced by cream. Milk, however, is much less deleterious to a diabetic patient than might have been supposed. In a girl with confirmed diabetes I made the following trial of the effect of milk. For four weeks she was fed on animal flesh and bran cakes; during the succeeding four weeks three pints of milk daily were added to this diet; and for three weeks subsequently, the milk was withdrawn. The annexed table shows the exact results of the treatment.

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[1] Prof. Cantani, of Naples, advocates a more extreme degree of non-saccharine and non-amylaceous diet than any previous writer. He gives his patients absolutely, nothing but flesh and fish—forbidding even eggs, green vegetables, and milk. In the way of beverages he forbids all wines, beer, tea, and coffee, and allows only simple water or water with a slight admixture of rectified spirit, or lactic acid. He claims for this rigorous treatment much more complete success than has hitherto been attained by the restricted diet as ordinarily prescribed. His experience, however, is limited to five cases. *Il Morgagni*, 1870.

	Average daily quantity of urine.	Average quantity of sugar daily excreted.	Increase of weight.
Meat diet, and bran cakes; for four weeks	. 55 oz.	897 grains.	5 lbs.
Meat diet, bran cakes, and three pints of milk; for four weeks	. 49 oz.	1260 grains.	5 lbs.
Meat diet, gluten bread, and cabbage; for three weeks	. 41 oz.	1020 grains.	7 lbs.

The patient continued to gain weight and to improve in her general condition under the use of milk, although the density of the urine and the excretion of sugar somewhat increased. A limited supply of milk may therefore be allowed.

Liver, as found in the butcher's shops, contains a considerable quantity of sugar; it also contains amyloid substance, which is changed into sugar by the saliva and pancreatic juice. Liver is therefore to be avoided by diabetic patients. The edible mollusks—oysters, cockles, mussels, etc.—are also improper, on account of the large quantity of amyloid substance contained in their enormous livers. For the same reason, the "pudding" of crabs and lobsters is objectionable.

The prohibited articles among vegetables are much more numerous and important, and the substitutes less perfect and more difficult to find.

The oldest substitute for bread is the "bran cake." The husk or bran of wheat consists of lignin and an albuminoid substance, and is quite devoid of starch. When this is washed and ground it may be made up into a rude imitation of bread with butt and eggs, and constitutes a valuable addition to the diet of a diabetic patient.[1]

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[1] The best formula for bran cakes is the following, supplied by Dr. Camplin: "*Formula for Bran Cakes.*—Take a sufficient quantity (say a quart) of wheat bran, boil it in two successive waters for a quarter of an hour, each time straining

Another important substitute is Bouchardat's "gluten bread." This is prepared by washing out the starch from wheaten flour, and working up the remaining gluten into loaves and cakes. This bread is manufactured on a large scale in France, with the aid of powerful machinery for inflating the dough with compressed air, or carbonic acid gas. It forms a light and elegant, and by no means unpalatable bread. Gluten is also ground down into a meal, and may be used for thickening broths and making puddings.[1] These preparations are not quite free from starch; all the samples examined by me showed an intense blue coloration with iodine.

By far the most palatable form of gluten bread is that prepared by Mr. Bonthron, of 106 Regent Street, London. He sends it out in the form of small buns, which eat crisp, and keep about a fortnight. Those of them that I have tested were nearly free from starch.

Dr. Pavy has introduced rusks and biscuits prepared from the starchless meal of the sweet almond.[2] These are more expen-

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it through a sieve, then wash it well with cold water (on the sieve), until the water runs off perfectly clear; squeeze the bran in a cloth as dry as you can, then spread it thinly on a dish, and place in a slow oven; if put in at night let it remain until morning, when, if perfectly dry and crisp, it will be fit for grinding. The bran thus prepared must be ground in a fine mill and sifted through a wire sieve of such fineness as to require the use of a brush to pass it through; that which remains in the sieve must be ground again until it becomes quite soft and fine. Take of this bran powder 3 oz. (some patients use 4 oz.), the other ingredients as follows—three new-laid eggs, 1½ oz. (or 2 oz. if desired) of butter, and about half a pint of milk, mix the eggs with a little of the milk, and warm the butter with the other portion; then stir the whole well together, adding a little nutmeg and ginger, or any other agreeable spice. Bake in small tins (pattipans), which must be well buttered, in a rather quick oven for about half an hour. The cakes, when baked, should be a little thicker than a captain's biscuit; they may be eaten with meat or cheese for breakfast, dinner, or supper; at tea they require rather a free allowance of butter, or may be eaten with *curd* or any of the soft cheeses.

"It is important that the above directions as to *washing* and drying the bran should be exactly followed, in order that it may be freed from starch, and rendered more friable. Mr. White, of Holborn, who made my mill, and was subsequently employed by others, attempted to grind the bran for them, and failed, from not washing and drying the bran, which, in its common state, is soft and not easily reducible to fine powder. In some seasons of the year, or if the cake has not been well prepared, it changes more rapidly than is convenient. This may be prevented by placing the cake before the fire for five or ten minutes every day."—(Camplin on Diabetes—Appendix.) These cakes may be had from Blatchley, 362 Oxford Street, London. The mills for grinding the bran are made by Gallop, 119 Cheapside.

A diabetic patient of mine says the cakes are much improved by using seven instead of three eggs. The addition of a teaspoonful of bicarbonate of soda is also an improvement.

[1] Gluten bread and other gluten preparations made after Bouchardat's formula, are supplied by Van Abbott & Co., Howford Buildings, Fenchurch Street, London. They may also be had of Jewsbury & Brown, Market Street, Manchester.

[2] Almond rusks and biscuits are supplied by Hill, Bishopsgate Street, London.

sive than the foregoing; but I have found that patients relished them as a change.

None of these substitutes is as palatable as ordinary bread: but they are of great service; and may be used one after the other, as the patient's inclination may direct. When none of these can be had, or when the patient refuses all three, as is sometimes the case, resource may be had to "torrefied" bread. Thin slices of ordinary bread are toasted before the fire until they are deeply and thoroughly browned—almost blackened. The starch and gluten are in great part destroyed by the heat, but the hungering diabetic relishes greatly the charred remnants when well buttered and eaten with other articles.

Rice, tapioca, sago, semola, macaroni, and vermicelli, all contain great abundance of starch, and are therefore inadmissible. Apples, pears, gooseberries, currants, plums, oranges, and all sweet fruits, are likewise pernicious from the quantity of sugar which they contain.

In place of potatoes, turnips, carrots, parsnips, beans, and peas—all of which contain starch or sugar—substitutes may be found in green vegetables—cabbage, endive, spinach, broccoli, Brussels sprouts, lettuce, spring onions, watercress, mustard and garden cress, and celery.

There does not seem to be any real advantage in forcibly curtailing beyond a moderate degree, the fluids taken by diabetic patients. The volume of the urine and the separation of sugar may be temporarily reduced by this means, but the general distress increases, owing to the more intense impregnation of the blood with sugar. Prout recommends that all fluids be given in a tepid state, as they thus allay the craving for liquids more effectually than when taken cold.

In the way of beverages, tea and coffee (without sugar) may be used. Chocolate made with gluten meal, and soda water, may also be used. The free use of wine and spirits, which is especially recommended by Bouchardat as a part of the diabetic regimen, is of more than doubtful propriety. Exact observations do not support Bouchardat's views, which are based on theoretical grounds. Griesinger found that the use of red wine, to the extent of a bottle and a half or two bottles per day, strengthened with two ounces of rectified spirit, increased considerably both the quantity of urine and the excretion of sugar. In a second observation by the same author, the use of alcoholic drinks caused, in addition to the above effects, a copious diaphoresis of saccharine sweat. The observations of Garrod, Camplin, Rosenstein, Siemssen, and Heller, are also unfavorable to the free use of beer, wine, and spirits. They should therefore be used sparingly. The best are those which are most free from sugar, namely, dry sherry, claret, bitter ale, brandy, and whiskey. Those to be avoided are port, sweet and effervescent wines, sweet ales, porter, rum, and gin.

The use of acid drinks, and especially dilute phosphoric acid, has been highly spoken of in some quarters. Griesinger reports unfavorably of their effects. He pushed dilute phosphoric acid to the extent of an ounce daily. At first, and under the smaller doses, the patient seemed to do very well; but after ten days, and with the full quantity, the volume of the urine and the proportion of sugar slightly increased, and the general state of the patient grew worse. I have frequently employed bitartrate of potash water for the purpose of allaying thirst, with good effect.

The patient should be clad from head to foot in flannel, in order to encourage the action of the skin, and defend the patient from the chilly sensations so common in this complaint. A warm bath once or twice a week is also very grateful to the patient, and abates the harsh, arid condition of the skin.

The results obtained from the dietetic treatment differ a good deal, according to the intensity of the disease, and the length of time it has existed. The following records illustrate the varying degrees of amendment which may be anticipated in confirmed cases: In the first two cases the patients were permanently cured. The third and fourth cases were inveterate, and, strictly speaking, incurable; in these the quantity of the urine was restored (temporarily at least) almost to its natural limits, and the patients gained flesh and strength in a very remarkable degree; sugar, however, still persisted in the urine, and any deviation from the prescribed regimen was sufficient to reawaken the diabetic symptoms in full intensity. In the fifth case, not much more than a temporary arrest of the downward course was attained, and this was speedily followed by a resumption of the untoward march to a fatal termination.

Case 1.—C. R., æt. 39, of a corpulent habit, came under my care in October, 1861. The urine amounted to eight pints a day; specific gravity 1040; it contained a large quantity of sugar. He had lost much weight, but was still in full flesh. The ordinary symptoms of diabetes were present in moderate intensity. C. R. had been dyspeptic for about fourteen years, though his habits had been, in every respect, temperate. He underwent the operation of lithotomy when a child. For the last two years he had perceived that he gradually lost flesh, had an unusual thirst and frequent desire to pass water. During this period, he had to get up three or four times each night to empty the bladder. Latterly the ankles had begun to swell. Most of the teeth were carious, and the gums loose and spongy. For two months the patient was treated as an out-patient of the Royal Infirmary, and enjoined to avoid saccharine and amylaceous articles of food. It was found that the treatment was carried out very inefficiently; he was therefore admitted as an in-patient on December 4, 1861.

For a week, he was abandoned to the ordinary mixed diet of the hospital. During this week he voided daily on an average 160 ounces of urine; specific gravity, 1035–1040; mean daily excretion of sugar, 5680 grains. He was then put on a diet of animal substances, with cabbage and bran cakes. In the week succeeding the change of diet, the mean daily discharge of urine fell to 60 ounces; specific gravity, 1022–1026. The sugar fell on the third day to 134 grains, on the fourth to 116 grains, and at the end of the week to 48 grains. In the second week the urine fell to its natural volume and density, and the sugar was reduced to a mere trace. This trace persisted for six weeks, when it suddenly disappeared. The patient gained weight at the rate of three pounds a week. He was then made an out-patient, and directed to continue the restricted diet.

A trace of sugar reappeared, from time to time, for seveval months, but ceased altogether in about eight months. He gradually resumed the moderate use of ordinary bread, and came to show himself at intervals. I saw him last in February, 1865. The urine was found perfectly free from sugar, and the general health and embonpoint were completely restored.

Case 2.—T. H., a very stout, florid-complexioned man, 34 years of age, who weighed, when in health, over sixteen stone, came under my care September 19, 1864. He stated that in the previous July, when the weather was very sultry, he perspired very freely, and drank large quantities of cold effervescing beverages.

From this period, a violent thirst and frequent desire to void large quantities of urine, tormented him. He lost weight to the extent of about 40 lbs.; he was voraciously hungry, and his strength gradually declined.

When first seen by me, the daily discharge of urine amounted to eight pints; specific gravity, 1048; sugar, 7540 grains per day. The general symptoms were mild. The tongue and skin were moist, the teeth sound, the gums only slightly spongy. He complained of incessant thirst, inordinate appetite, pain in the back, and feebleness.

He was put on a restricted diet on September 22d, and observed the directions given to him with the most praiseworthy strictness. He was allowed bran cakes, butter, fresh meat, eggs, cabbage, tea and coffee sweetened with glycerine *ad libitum*. He was cut off from potatoes at once, and, after two days, likewise from ordinary bread, and limited entirely to the articles above enumerated. A warm bath was administered every evening, and a pill containing half a grain of opium and one grain of sulphate of iron was given three times a day.

On the third day great improvement had taken place. The urine was reduced to 50 ounces; specific gravity, 1028; sugar, 210 grains.

For two days the patient's condition remained in every respect stationary; but on September 28th he did not feel so well. The urine had fallen to 20 ounces, and the sugar to a very small quantity; the pulse was 98, tongue furred, and a degree of pyrexia prevailed. He sweated profusely after the baths; and some hemorrhoids, to which he was subject, became very painful, the bowels being confined.

This disturbance was attributed partly to the somewhat too sudden revolution in the diet, and partly to the constipating effects of the pills.

On September 29th the pills were withdrawn, the baths were administered every other evening, instead of daily, a little ordinary bread was allowed, a dose of castor oil administered, and the patient directed to keep his bed.

In a few days this disturbance subsided, and the restricted diet was again rigidly enforced. Rapid amendment set in; the urine returned to its natural quantity and density; the sugar gradually diminished, and on October 17th it had entirely disappeared from the urine.

The restricted diet was adhered to for another fortnight, and then a gradual return to the use of ordinary bread was permitted, the urine being carefully examined for sugar from time to time, but none found.

At the beginning of 1865, the bran cakes were discontinued; ordinary bread was allowed freely, and a small portion of potatoes. At the end of January, all restrictions on the diet were withdrawn. The patient had now reached almost his original weight of 16 stone, and felt himself in every respect perfectly well. He was last seen on July 25, 1865. The urine was found perfectly free from sugar.

In the first of these instances, a confirmed but mild case of diabetes, of two years' standing, was perfectly and permanently cured by the dietetic treatment in about eight months. In the second instance, diabetes of three months' standing was completely cured in less than a month. Recoveries so complete as these are, unfortunately, rare. The two following are examples of the *conditional* amelioration, which may be commonly attained, even in severe cases:

CASE 3.—E. H., a well-grown girl of sixteen, a factory hand, had been diabetic for three years. She was admitted into the Manchester Infirmary, March 26, 1860.

The disease was uncomplicated, and exhibited in great severity the outward signs of diabetes in an advanced stage. There was a harsh dry skin; a tongue like a piece of broiled ham, and deeply furrowed; abdominal pains, constant drowsiness, consuming thirst, gross appetite. dry scurfy skin, and great emaciation.

For a fortnight after admission, she was put on the common diet of the hospital, which includes a liberal allowance of meat, potatoes, and bread. The state of the urine, during the last six days of this fortnight, was as follows: Mean daily discharge, 210 ounces; mean daily excretion of sugar, 10,400 grains; average density, 1042. Her weight was 80 lbs.

The diet was then changed. Milk and all vegetable compounds were withdrawn; instead, she was allowed an unlimited supply of eggs, fresh meat, and beef-tea. The patient did not, however, observe my directions strictly, but obtained, and surreptitiously consumed, certain quantities of oranges, sugar, and treacle-toffy. Nevertheless, a remarkable improvement in her condition took place. At the end of eleven days, the mean results since the change of diet were: Daily discharge of urine, 70 ounces; sugar, 1860 grains; average density, 1034. Weight, 81 lbs.

The general health was also much ameliorated; the skin was softer, the tongue less fiery, the thirst and appetite allayed.

On April 21st, bran cakes were added to the animal diet, and greatly relished by the patient. From this date to May 16th—a period of 26 days—no further change was made. The results are shown in the following table. I have divided the period into weeks, for the purpose of displaying the gradual progress:

	Average daily quantity of urine. Ounces.	Range of density.	Sugar each day. Grains.	Weight. lbs.
First week	54	1025–1033	1160	81
Second week	67	1021–1031	970	84
Third week	51	1022–1035	870	85
Fourth week	49	1019–1035	690	86
Entire period	55	1019–1035	897	

With this increase of weight, her general condition had improved; the tongue had become pale and moist, but it was still mapped on the surface, and unnaturally smooth.

On the 16th of May, milk was added to the previous diet; the results are given in a preceding page (*see* p. 273). On June 12th, milk was again withdrawn, and gluten bread substituted for bran cakes. Cabbage was also allowed with dinner. The flow of urine on this diet averaged 41 ounces, and the sugar 1020 grains per day. The body-weight went on increasing to 98 lbs. Her general condition was now, at the end of eleven weeks of treatment, such, that an unprofessional person would have pronounced her cured. The outward signs of diabetes had disappeared; the skin was restored to its natural softness; the thirst and appetite were no longer inordinate; the flow of urine was reduced within the normal compass. The patient had gained 18 lbs. in weight; she slept soundly, had neither pain nor ache; her strength was so far restored, that she was able actively to assist the nurses in the work of the wards. She came from a distant town, and her history after leaving the Infirmary is unknown to me.

CASE 4.—W. A., a factory hand, æt. 30, was admitted as an out-patient, October 12, 1859. He presented the usual appearance of diabetes in full career. The disease was uncomplicated, and had existed about a year. The quantity of urine varied from 10 to 15 pints daily, and its density averaged 1044. The patient was directed to observe a restricted diet; and a pill containing a grain of opium, with a quarter of a grain of sulphate of iron, and half a grain of quinine, was ordered three times a day. This treatment was continued—the doses of opium being gradually increased—for seven months. A marked improvement took place; the diabetic symptoms abated considerably; the tongue became moist; the urine fell to five or six pints daily, with a specific gravity of 1040. The sugar averaged 4400 grains. He gained strength and some weight, and was able to resume his occupation for a time. As his condition appeared stationary, he was made an in-patient on May 8, 1860. On his admission, all medicines were discontinued, and the patient was allowed the mixed diet of the house. The effect of this change was a sudden reappearance of all the untoward symptoms, with a sense of great debility, and an alarming cough. The condition of the urine was as follows: Daily discharge of urine, 205 ounces; sugar, 7400 grains; average density, 1042. Three days of this freedom from treatment had forced him to keep his bed.

I now gradually withdrew all amylaceous substances, and substituted meat, fish, eggs, and beef-tea. He was also allowed eight ounces of brandy daily. After the change was completed, the diet was absolutely devoid of starch and sugar. Under this diet, the urine altered greatly for the better. During the first week of the restricted diet, the daily discharge of urine was 61 ounces; daily excretion of sugar, 928 grains; average density, 1032. The general symptoms also improved, but not in proportion to the amelioration in the condition of the urine.

A second week of the same treatment brought down the urine to: Daily discharge, 56 ounces; daily excretion of sugar, 658 grains; average density, 1028.

I was now met with the difficulty which so many have encountered in pursuing this treatment; namely, a total failure of the appetite, and consequent alarming depression of all the vital powers. To obviate these untoward events, the patient was allowed bran bread and the free use of green vegetables—cabbage, lettuce, and watercresses. A grain of opium was also given three times a day. The diet was therefore still starchless, and almost entirely devoid of sugar. Decided amendment followed this change, and in a few days the returning strength and cheerfulness kept pace with the improved appetite and increasing weight.

During the remainder of his stay in the infirmary, a period of two months, no further change of importance was made in the diet or medicine. The patient's weight on admission was 97 lbs.; but it rapidly sank in the first few days, and at the end of three weeks it was only 91 lbs. From this time onward, however, the weight began to increase, and it went up gradually to 105 lbs., which point it had reached the week of his discharge.

The state of the urine for the last two months was remarkably constant. The daily discharge varied from 40 to 60 ounces; the daily excretion of sugar, from 800 to 1000 grains; the average density, from 1030 to 1033.

The excretion of sugar ruled higher than when the diet was exclusively animal. This I attributed to the improved appetite, which enabled the patient to take more nourishment, rather than to any untoward influence exercised by the green vegetables.

I might greatly multiply examples of this class; but it will be more useful to illustrate the less fortunate results for which the practitioner must also be prepared.

CASE 5.—E. B., a niece of the patient C. R., who made so good a recovery, was admitted into the Royal Infirmary in December, 1862. She had been diabetic for 16 months; and suffered from excessive thirst, voracious appetite, and great emaciation. The tongue was glazed, skin harsh and dry. There was no complication. The urine amounted to 15 pints a day, and contained over 10,000 grains of sugar, when she lived on a mixed diet.

She remained in hospital two months; and was gradually limited to a diet of animal flesh, with eggs, cabbage, and bran bread. On this diet she slowly gained three pounds in weight, and improved sensibly in her general health. The urine, however, never fell below five pints; usually it oscillated between seven and eight pints, with a specific gravity ranging from 1030 to 1040, and a daily excretion of sugar of 4450 to 7420 grains.

After leaving the Infirmary, she speedily relapsed, gradually grew worse, and died in March, 1863, in the Withington Workhouse.

Much discredit has been thrown on the dietetic treatment, by a slovenly and incomplete manner of carrying it out. It requires most vigilant watching to keep guard against the admission of forbidden articles. The patient's own craving for them is often too much for his resolution, and most artful deceits are practised on the medical attendant. This is especially the case at the beginning of the treatment. After awhile, the patient perceives, from his own experience, the importance of abstaining, and the desire for the forbidden articles diminishes very notably after the lapse of some weeks. Amylaceous compounds, too, are often unwittingly administered by the attendants. Starchy matter is never absent from the cook's hand; it enters, in one guise or other, into almost every dish.

Then there arises the other difficulty—the rejection by the stomach of the restricted diet. This difficulty is, perhaps, made too much of. A skilful selection and frequent change of articles of diet, usually suffice to reconcile the digestive organs. The field of selection among admissible articles is so wide that, in private practice, the practitioner's resources are almost inexhaustible. Among hospital patients, however, the embarrassments on this score are very serious.

There are cases of such severity, that not even a temporary amendment can be obtained by the dietetic treatment. I have known more than one such instance in children under ten years of age, in whom the disease ran a rapid course, and proved fatal in a few months. There are also a certain number of chronic cases in which the dietetic treatment proves unsuitable, and hastens rather than retards the final catastrophe. These are, for the most part, long-standing cases—cases, perhaps, which have been benefited at a previous epoch by that treatment. In two of my Infirmary patients, who were readmitted to the benefits of the charity after an interval of several months, a much more decided amelioration followed the dietetic treatment during the first period of their stay, than during the second.

The sugar-forming vice of the diabetic system appears at first (and throughout in the milder cases) confined to saccharine and amylaceous articles of food; but as the disease becomes inveterate, the assimilation of the albuminous principles is more and more affected, until, at length, these yield sugar almost as readily as the former. Griesinger found in a case of this kind, on strict flesh diet, that three-fifths of the albuminous materials reappeared in the urine as sugar. When matters have come to this pass, it is not to be wondered at, that the patient no longer derives benefit from a restricted diet, which he can only use sparingly, and almost with disgust, and that he should, on the whole, find himself in a better position when abandoned to ordinary mixed fare, which he can consume in abundance, and with relish. Experience is imperative on this point. When a flesh diet, judiciously eked out by appropriate substitutes for bread and potatoes, fails to ameliorate the general condition, it should not be too obstinately persisted in after a fair trial. The practitioner should give way first with regard to bread, and hold out longest against potatoes. No inflexible and universal rule can be laid down respecting the diet of diabetic individuals, under all circumstances and in all stages of the complaint. Cases will occur, in which the power to take a plentiful supply of a mixed diet, more than compensates for the increasing thirst and freer discharge of urine and sugar. I have also noted that some of the milder types of this disease in which saccharine urine is unaccompanied with diuresis, are made worse by a too restricted diet. (*See* Appendix to this chapter.)

Dr. Donkin[1] recommends another mode of carrying out the animal diet treatment—namely, by putting the patient on a diet exclusively composed of skimmed milk, which he administers in quantities of six or eight pints daily—persevering rigidly with the treatment, if necessary, for ten or twelve weeks. I have seen several patients who tried this severe method. Few of them could tolerate it except for a few days—and those who continued longer were rapidly reduced. Three chronic cases I know of, in which the treatment was obstinately persevered with, ended in fatal exhaustion. One of them had been under my care for a considerable period, and the patient maintained a fair state of health under a moderately restricted diet and the use of opium—three months of the skimmed milk treatment brought the case to a fatal termination.

MEDICINAL SUBSTANCES IN DIABETES; SUPPLEMENTARY MEANS.—Some of these are employed under the impression that they possess a really curative power in this disease; others are resorted to simply as adjuvants, to combat some particular symptom.

The inquiries hitherto made on the supplementary means—medicinal and other—employed in the treatment of diabetes, are mostly vitiated, by an insufficient separation of their effects, from those of the restricted diet, which is usually conjoined therewith, and a want of attention to the clinical grouping of the cases. A number of remedies have been extravagantly lauded on diverse hands, and have in this way attained an ephemeral reputation; but when tried by accurate observers, they have proved to be absolutely inert. Unless the points just indicated are kept in view, only misleading conclusions can be drawn from any inquiries on this subject. It is quite possible, that remedies which have proved powerless in inveterate cases may be of real service in milder examples of a different type, or in the earlier stages of the disease. A complete revision of the supplementary means of treating diabetes is loudly called for. It may be taken for granted, in the present state of knowledge, that the general basis of all treatment of diabetes must be the dietetic restrictions already described. Other means should be studied with a clear understanding of their supplementary and subordinate place.

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[1] On the Milk Treatment of Diabetes and Bright's Disease. London, 1871.

OPIUM.—This narcotic is unquestionably of great use in the treatment of diabetes—not from its direct influence on the course of the disease, but from its anodyne properties. If no restriction be placed on the diet, opium in doses of from 6 to 20 grains a day has always, in my experience, had the power of reducing the flow of urine by about one-half; that is to say, of bringing it down to five or eight pints, and this without increasing its density. But, notwithstanding this amelioration in the state of the urine, the downward progress of the disease is not arrested; and the effect of the drug seems attributable to its deadening influence on the appetite, rather than to a specific power of checking the formation of sugar. When opium was given to patients under a restricted diet, it did not in my hands exhibit the least power of lessening the flow of urine or the excretion of sugar. Its value depends on its power of inducing sleep, and of allaying the dolorous sensations and irritability which constantly torment diabetic patients.[1]

There is a great tolerance of opium in confirmed diabetes. Doses of 2, 3, and 5 grains, three times a day, are generally borne without the production of any appreciable narcotism.

Dr. Pavy prefers *codeia* to opium. He finds that this alkaloid yields all the good effects of the crude drug without some of its disadvantages. He gives it in gradually increasing doses, from a quarter of a grain to two grains three times a day. Other observers have also recommended codeia, but I cannot say that my experience altogether justifies this preference.

ALKALIES.—Alkaline substances have been specially recommended by Miahle, on account of their supposed power of favor-ing the oxidation and destruction of sugar in the blood. These theoretical views are now overthrown In two of my patients, I made a trial of full doses of the bicarbonate of potash. One of them was on a mixed ordinary diet, and the disorder was far advanced. The urine was rendered alkaline for ten days without in any way altering the excretion of sugar, or the general condition. In the second case, the patient was on a restricted diet. She took for a fortnight 320 grains of the bicarbonate daily, in divided doses; the urine was thereby rendered freely alkaline. During the week preceding the alkaline treatment, 1160 grains of sugar were excreted daily. In the first week of the alkaline treatment 970 grains a day were separated, and in the second week 870 grains. In the week following the withdrawal of the alkali, the sugar amounted to 690. This observation tends to show that the alkali had no appreciable influence on the excretion of sugar. I have not encountered any difficulty in rendering the urine alkaline in diabetes, as Dr. Pavy seems to have done.[1]

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[1] *See* the author's paper—Brit. Med. Journ., 1860.

Rennet and *Pepsine* have been vaunted in such terms of confidence, as to raise hopes which are not destined to be realized. The most remarkable results, obtained from rennet, are those published by Dr. James Gray. He states that of twenty-seven persons treated seven recovered. This is an example of most rare success, and it is to be regretted that the cases are not reported with that exactitude and detail which are desirable on such debatable ground. In all of them a rigid adherence to animal diet and bran bread was insisted on; and it seems more than probable that the amendment in each case was due to the restricted diet rather than to the rennet. Dr. Nelson, of Birmingham, extols the same remedy. His cases do not seem to have been severe ones; and the diet was regulated in at least some of them. The reports are much more imperfect than those of Dr. Gray.

I gave rennet a resolute trial in one confirmed case. It was prepared in the manner recommended by Dr. Gray, and given in doses of two tablespoonfuls three times a day. The patient took it for more than two months, conjoined with a rigidly restricted diet. During this period he improved, and gained 5 lbs. in weight. But he was improving just as rapidly before he began the rennet, and the daily excretion of sugar had not in the least diminished during its use. Griesinger, in two cases accurately observed, found even a slight increase of sugar during the use of rennet. Other trustworthy reports are equally unfavorable.

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[1] The alkaline and ammoniacal phosphates and the carbonate of ammonia have again been tried by Basham and Pavy, and the citrate of soda by Guyot. *See* Brit. Med. Journ., 1869, i. 323; and *ibid.*, p. 590; and Syd. Soc. Year Book, 1865–6, 70. Recently the use of ammonia in the form of carbonate and acetate has been strongly recommended in diabetes. (Adamkiewicz, Pflüger's Archiv., 1879, p. 160.) The evidence in its favor is as yet, however, scanty.

Parkes[1] and Leubuscher[2] found pepsine useless.

I conceived that it was worth a trial, whether some of the substances which act powerfully on the nervous system, might not exercise a beneficial effect in diabetes. With this view, I exhibited strychnia and belladonna, in gradually increasing doses, until their physiological effects began to be perceived. But not the slightest influence on the excretion of sugar could be discovered during their use.

Among the more recent remedies employed in diabetes may be mentioned arsenic,[3] iodine, bromide of potassium,[4] picric acid, and Calabar bean. The last three I have tried and found useless. The evidence in favor of the first two is too slender to excite much hope. I have tried both peroxide of hydrogen and ozonic ether, only to find that the hopes held out with respect to them were altogether delusive.[5] Salicylate of soda has been used by several physicians, who have reported very good results from its administration.

I have given lactic acid, as recommended by Cantani, repeatedly and resolutely, without seeing the slightest advantage from its use.

Independently of so-called specific remedies, there is a large field for the skilful use of adjuvant means, employed simply for their ordinary therapeutical effects. The obstinate constipation which commonly prevails in diabetes, must be corrected by a regulated use of castor oil, seidlitz powders, or the ordinary rhubarb and magnesia mixture. Anodynes are called for to subdue pain, nervous exhaustion, restlessness, and insomnia. Dyspeptic symptoms are to be combated by alkaline tonics: and for this purpose I know of no better combination than the bicarbonate of potash in infusion of calumba, with hydrocyanic acid. The poverty of the blood and the progressive emaciation are best combated by long courses of iron and cod-liver oil. I have already spoken of a solution of bitartrate of potash, as the best means of directly allaying the thirst. When the craving for food, and sense of sinking at the epigastrium are troublesome, a pill containing two or three grains of assafœtida, administered twice or thrice a day, often gives most striking relief.

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[1] On the Composition of the Urine, p. 356, note.

[2] Arch. f. Path. Anat., Bd. xviii. 119.

[3] Bulletin de Thèrap., 1870, xlvi. 519; Berlin. Klin. Wochensch., No. 12, 1869; O. Rees, Lancet, 1864, ii. 436; and Leube., Archiv Gén., 1870, p. 602.

[4] Austin Flint, Amer. Journ. of Med. Sci., 1870, 282; and Begbie, Edin. Med. Journ., xii. 487.

[5] Moleschott has recently recommended iodoform, and has obtained good results from its use. (Wiener. Med. Wochensch. No. 17, 1882.)

Diabetic patients often reap considerable benefit from change of air, and a sojourn at watering-places. The Bristol Hotwells, Vichy, and Carlsbad waters have obtained some celebrity for their utility in diabetes. Dr. Murrell has recommended Bethesda water. In milder cases, sea-bathing may be recommended in moderation in the hot season of the year.[1]

Saccharine Treatment of Diabetes.—Piorry conceived the odd idea that the main evils of diabetes depended on the loss of sugar through the kidneys, and that, by compensating this loss by administering sugar internally, these evils could be overcome. Dr. W. Budd, of Bristol, followed up Piorry's lead, and administered 5 to 8 ounces of sugar and 4 ounces of honey to two diabetic patients, with great benefit. Ordinary mixed diet (including potatoes) was conjoined. These results provoked new trials of this treatment by Dr. Burd, of Shrewsbury, Dr. Sloane, of Leicester, Dr. Bence Jones, and Griesinger, but with results so decidedly unfavorable as to leave no doubt of the inutility of the practice. A full *résumé* of the results of the saccharine treatment of diabetes may be found in a paper by the author in the "Brit. Med. Journ." for November, 1860.

APPENDIX.

Milder Types of Diabetes.

The cases brought together under this heading are somewhat miscellaneous; and they do not present those marks of uniformity, which are required to constitute a homogeneous pathological group. They are separated from classical diabetes by certain broad distinctions of clinical importance; but they exhibit among themselves certain disagreements which make it evident that they represent more than one type of disease.

From ordinary or classical diabetes, these milder types are distinguished by all or some of the following signs: Absence of a fixed tendency to a fatal termination; absence, or only moderate degree of thirst, voracity, and emaciation; slight or temporary increase in the quantity of urine; transitory duration; amenability to treatment; slight, moderate, or intermittent glycosuria.

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[1] Bouchardat speaks in high terms of enforced exercise and gymnastics for diabetic patients. See Annuaire de Thérap., 1865, p. 291.

The greater number of these cases fall within one or other of the three following groups, to each of which illustrative examples are appended:

GROUP I.—Urine persistently saccharine; density 1030 to 1043; diuresis absent, or very moderate; no excessive thirst or appetite; moderate conservation of strength and flesh; stationary condition.

CASE 1.—Mr. B., a manufacturer, æt. 45, thin but not markedly emaciated, able to attend to his business, consulted me May 14, 1861. His health had been feeble for six months. He complained of weakness, loss of appetite, and restlessness. The urine had never exceeded four pints, and usually did not exceed three pints in the twenty-four hours. The specimen sent to me for analysis had a density of 1042, and contained 7.2 per cent. of sugar. There was no inordinate appetite or thirst; the skin was moist. The patient had tried a diet composed of animal flesh and green vegetables, but had been unable to adhere thereto on account of the total failure of the appetite.

During the last four years I have seen this patient several times. His condition continues unchanged, both as to the general health and the state of the urine. He is still a valetudinarian, but goes about his business, and observes a diet, only restricted with respect to the use of potatoes.

CASE 2.—Mr. F., æt. 50, formerly engaged in business. He consulted me in November, 1862, and stated that he had been ailing about three years, suffering from indigestion, lowness of spirits, and loss of strength. A twelvemonth before sugar had been detected in the urine. The urine had not at any time exceeded three pints in the twenty-four hours. He has never been troubled with thirst; the skin is usually moist; there has been slight emaciation. He has tried a restricted diet without any benefit.

Two specimens of urine were handed to me for analysis; one on November 20, 1862, and the other on April 22, 1863. The former contained 7.7 per cent. of sugar, and the latter (which had a specific gravity of 1039) 6.3 per cent. The daily quantity at both dates was three pints. The disorder in this instance appears to have arisen from worry and anxiety connected with business; but for a period of two years after giving up business he remained *in statu quo*, no treatment appearing to have any beneficial result. Recently he has been in much better health, has recovered his weight, strength, and cheerfulness, and believes himself thoroughly rid of his complaint; and yet, the urine has now (February 23, 1865) a specific gravity of 1035, and contains 5.7 per cent. of sugar. In the autumn of 1870 I was called to see this patient again. The sugar had now for a long time disappeared from the urine and was replaced by albumen. He was suffering from general anasarca, and all the other signs of chronic Bright's disease—of which he died in December, 1870.

CASE 3.—Dr. Latham[1] relates a case resembling these in most respects. The patient was a gentleman, æt. 40, well known in the profession of the law. The urine at no time exceeded a quart, but it was so sweet "that it might easily have been mistaken for syrup." The dietetic treatment was resolutely tried without any good effect: he died with cough, colliquative sweats, and other signs of phthisis.

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[1] Latham on Diabetes, p. 147.

GROUP II.—Glycosuria, temporary or intermittent; thirst and diuresis moderate, or none; little emaciation and loss of strength; the complaint depending on mental anxiety, blows on the head, or concussion of the spine, and terminating in complete recovery.

CASE 4.—A gentleman, æt. 46, engaged in business, consulted me on March 23, 1862. He had suffered from slight, recurrent, dyspeptic symptoms for more than a year, together with numerous nervous phenomena and loss of rest. During this period he had undergone great mental stress in connection with the responsibilities of a large manufacturing concern. On two occasions he had been seized with some kind of fit, which, from the description given, appeared to be a bastard epilepsy. In one of these he had fallen from his horse; but there was no direct injury to the head. At my request a specimen of urine was sent for examination. Its specific gravity was 1035, and it contained 5.2 per cent. of sugar; no albumen or other abnormal ingredient was present. The daily quantity did not exceed three pints. He was put on a moderately restricted diet, and recommended to make arrangements which would relieve him of a large portion of his responsibilities. He continued under my observation for six months. The sugar disappeared in about six weeks, except a trace, which also vanished at the end of four months. His health is now (July, 1865) perfectly restored.

One of the most singular instances of glycosuria, persisting for several months, unaccompanied with any of the symptoms of true diabetes, is related by Griesinger (loc. cit. p. 51).

CASE 5.—A medical student had, during a course of chemical instruction, in the year 18—, often examined his urine, and found it in every respect normal. He spent the summer of the succeeding year in Switzerland, and underwent a number of wettings on botanical excursions. Some months later, while in perfect health, the appearance of the urine attracted his attention. On examination it gave an abundant sugar reaction with Trommer's test. He now examined the urine daily, and found the density to vary between 1022 and 1027. The glycosuria persisted throughout the following winter, during which he continued to reside in the same moist and foggy locality. In the succeeding spring, Herr —— returned from Switzerland, and, being much occupied, had no longer any time to bestow on his diabetes; and when, in the course of the ensuing summer, he examined the urine again, he found it totally free from sugar, nor has a trace been found in it since. During the entire period that the urine contained sugar, he did not experience a single one of the known symptoms of diabetes.

GROUP III.—Glycosuria in persons advanced in years; of full habit; moderate conservation of flesh and strength; moderate diuresis; moderate amount of sugar; abundance of uric acid deposits; often gout; sugar sometimes present for years, varying greatly in quantity, sometimes intermitting—termination variable.

Dr. Bence Jones has published an account of a number of cases of this class.[1] Of twenty-nine cases of glycosuria, observed by him in the preceding three years, eleven were above sixty years of age, and six of these were above seventy. He supplies the following analysis of these eleven cases:

In 2, the disease was intermitting.

In 6, the quantity of urine was scarcely, if at all, increased.

In 1, the quantity was increased, but the disease had probably existed for sixteen years.

In 1, the urine was albuminous, and the diabetic symptoms were very slight.

In 1 (about seventy-four years of age), the disease existed in its intensity.

In all cases save one, the disease was of exceedingly mild character.

Many cases of this kind have come under my notice, of which the two following examples may serve for illustrations:

CASE 6.—Mr. A., a surgeon, æt. 60, a tall, stout man, of powerful frame, consulted me June 11, 1863. He had noticed for the last four months an undue frequency of micturition, with a certain languor unusual to him, of which, however, he thought little, until the copiousness of the urine excited his suspicions, and induced him to test it for sugar. This led to the detection of his complaint. He had lost some flesh.

When I examined him, he had a ruddy complexion and an appearance of health; the appetite was moderate; thirst somewhat troublesome; skin moist; he went about his usual business—being in extensive practice in a rural district—with scarcely more fatigue than ordinary. The teeth were extensively decayed. The urine amounted to five or six pints daily. A specimen carefully collected for twelve hours was sent to me for examination. It amounted to 68 ounces; specific gravity, 1034; it deposited uric acid copiously, and contained 6 per cent. of sugar, which indicated a total of 1800 grains in half a day.

Mr. A. was gradually put on a restricted diet, with gluten bread. In a week, the urine of twelve hours had come down to 45 ounces; specific gravity, 1035; percentage of sugar, 6.1; sugar voided in twelve hours, 1190 grains.

Four weeks later, the urine of twelve hours had diminished to 37 ounces; specific gravity, 1028; sugar, 4 per cent.; quantity voided in twelve hours, 673 grains. The general condition had also greatly improved; he still adhered to the restricted diet.

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[1] Med.-Chir. Trans., vol. xxxvi.

I have seen Mr. A. from time to time up to the present date (February, 1865). He is now perfectly restored to his original health and embonpoint. The restrictions on his diet have long since been relaxed. He derived considerable advantage from the use of almond rusks and cakes, and from change of air and scene, in the highlands of Scotland.

November, 1871.—Mr. A. died suddenly two years ago from a ruptured aortic aneurism.

CASE 7.—Mr. M., a retired solicitor, æt. 72, consulted me October 17, 1863. He was a florid-complexioned, stout, healthy, and vigorous-looking man for his age. Until sixteen months ago he had always enjoyed excellent health.

Sixteen months ago he was seized with a low febrile complaint of undetermined character. He kept his bed for two months, and was greatly reduced by it; but he gradually recovered, and went to Buxton to complete his convalescence. Before going to Buxton he had noticed a sweet taste in his mouth and a certain sweetness of the skin of his hands; and when there he noticed a great thirst and frequent calls to void urine. With the continuance of these symptoms he became rapidly thinner, and sent for his son-in-law, Dr. H., who examined the urine, and discovered sugar. Dr. H. found the symptons of diabetes present in moderate intensity; gums spongy; emaciation very considerable; all his embonpoint gone; he was "reduced to a little old man." The urine amounted to six and ten pints a day; and his thirst was so tormenting, that he used to prepare for himself a large jugful of oatmeal-water and milk, to drink at night.

At this period, he was put on a strict flesh diet, with green vegetables. Great benefit followed this treatment; and in about two months from the first onset of the diabetic symptoms, he had recovered from the attack, and begun to recover flesh and strength. It was not ascertained whether the sugar disappeared from the urine when the other symptoms subsided.

He continued in improved health for five or six months, and regained much of his previous vigor. He then began to suffer from severe lancinating pains about the base of the chest. On account of these he sought my aid.

He complained of intense pain, of neuralgic character, along the course of the lower intercostal nerves. Up to the day before his visit to me, the pain had been limited to the left side, but it had now invaded the right side; and a painful circle embraced him, in a line corresponding to the attachments of the diaphragm. The pain was darting, burning, as if a red-hot iron was drawn round him; it prevailed in paroxysms; but lately the remissions had never been complete; and the pain came forward to the mesial line, and descended into the testicles and penis. Nightly opiates were required to induce sleep. He was very nervous and agitated, especially during the paroxysms, but there was no fever. Tongue clean, pulse quiet, ranging from 65 to 80 (he often counted it himself); heart's sounds were healthy, and there was no hypertrophy. The pain was much increased by motion of the body, as in walking. There was no thirst; the quantity of urine was not increased. Micturition frequent at night; appetite pretty fair.

At my request he brought me the urine made after dinner on October 18th; its specific gravity was 1030, clear, amber-colored; it contained no trace of albumen, but as much as 5.1 per cent. of sugar. He was ordered 5 grains of quinine, with some carbonate of iron, and a few drops of laudanum at night.

October 19th.—He brought me the urine made before breakfast; its specific gravity was 1019, and it contained only a trace of sugar. He had passed a much better night than usual.

20th.—Urine before breakfast contained a trace of sugar; that voided after dinner contained a good deal more.

21st.—Urine before breakfast was quite free from sugar; that after dinner contained 4 per cent. He still complained of the pain round the chest, but in much diminished degree.

25th.—Urine before breakfast free from sugar; that passed after dinner contained only 0.8 per cent.

He was put for a while on a partially restricted diet. The urine continued for some days to show traces of sugar after dinner. After this he left town and went to the country, continuing to improve. This gentleman is now (February, 1865) in very fair health for his age; but I cannot state whether or not the urine contains sugar.

In patients of this class I have generally found that, although the diabetic symptoms prove mild and amenable to treatment, life is seldom prolonged beyond a few years. The glycosuria may disappear or become insignificant; but the constitution is evidently broken, and they usually die in two, three, or four years, either from cerebral disease or from pulmonary complications.

CHAPTER III.

GRAVEL AND CALCULUS.

(*Urolithiasis.*)

GENERAL ETIOLOGY.

THE deaths from stone, in England and Wales, in the five years ending 1866, amounted to an annual average of 168. It is satisfactory to note that the mortality from this cause exhibits a progressive diminution in the last thirty years, as may be seen from the following table constructed from the Registrar-General's Reports:

Mortality from stone in England and Wales, in five successive quinquennial periods.[1]

In the 5 years	1838–42	the yearly average	of deaths	from stone was	297
"	1847–51	"	"	"	232
"	1852–56	"	"	"	216
"	1857–61	"	"	"	184
"	1862–66	"	"	"	168

The cause of this diminution is to be chiefly sought for in the earlier detection of the stone, and earlier resort to operation, in recent times; perhaps also in the improved diet and water supply of the population.[2]

Calculous disease is much more fatal (as might have been expected) in the male, than in the female, sex. For every female that died, in England and Wales, in the ten years, 1857–66, from the consequences of stone, nearly nine males perished.

More deaths from stone occur at an early *age*, and in the waning years of life, than in the intermediate periods, as is shown by the following table:

TABLE *showing the number of deaths from stone at different ages in the decade 1857–66 in England and Wales—Males only included.*

Under	5 years	116 deaths.
Between	5 and 15 years	114 "
"	15 " 25 "	59 "
"	25 " 35 "	62 "
"	35 " 45 "	78 "
"	45 " 55 "	132 "
"	55 " 65 "	294 "
"	65 " 75 "	517 "
"	75 and upwards	299 "

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[1] The returns of the years 1843–46 are tabulated differently from the remainder, and cannot, therefore, be included in this table.

[2] The suburban district of Hulme supplies considerably fewer cases of stone to the Manchester Infirmary since the pipe-water has replaced the old pump-water supply.

The great fatality of stone above the age of fifty-five is due, not so much to the greater frequency of stone at that epoch, as to its more severe effects on the constitution, and the less favorable results of operation in advanced life. The *frequency* of stone is far the greatest under five years of age; and next between ten and fifteen years. It then diminishes rapidly until the thirty-fifth year. Above this age cases of stone become, again, more and more frequent, until the age of sixty-five. The following table indicates, very exactly, the prevalence of stone at different periods of life. It embraces all the persons who underwent the operation of lithotomy, during given periods of time, at the following hospitals: Guy's, St. Thomas's, University College, Norwich, Cambridge, Oxford, Birmingham, Leicester, and Leeds.

TABLE *showing the ages of* 1827 *persons who underwent lithotomy at the above hospitals—constructed from statistics collected in Sir H. Thompson's work on Practical Lithotomy and Lithotrity.*

Under 5 years	473
Between 5 and 15 years	528
" 15 " 25 "	157
" 25 " 35 "	85
" 35 " 45 "	90
" 45 " 55 "	156
" 55 " 65 "	225
" 65 " 75 "	103
" 75 " 81 "	10

No countries or climates are altogether free from calculous disorders; but some localities are considerably more afflicted by them than others. Stone and gravel are common in England, France, Teneriffe, Iceland, and Egypt.[1] They are, on the contrary, rare in Sweden and Norway, Styria, and some other parts of the Austrian dominions. In Christiania, 3211 patients were treated in the general hospital during a period of four years, and among them there was only one stone case. In the hospital of Gothenburg, in Sweden, which contains sixty beds, not a single case of stone was received in fifteen years.[2]

The climatic conditions favorable to the prevalence of stone appear to vary within narrow topographical limits. Of the eleven registration districts into which England and Wales are divided, the eastern counties of Norfolk and Suffolk furnish the largest proportion of deaths from stone. Next to these come the North Midland counties. The fewest deaths from stone (as compared to the total mortality) are furnished by Lancashire and Cheshire, and by the Southwestern counties.[1]

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[1] The frequency of stone in Egypt is due to the ravages of the Bilharzia hæmatobia, a minute parasite which infests the urinary organs in hot countries. (*See* Bilharzia.)

[2] Civiale, Traité de l'Affection Calculeuse, p. 580.

CLASSIFICATION OF URINARY CALCULI, THEIR CHEMICAL CHARACTERS, ORIGIN, GROWTH, AND GENERAL CLINICAL HISTORY.

Urinary calculi may be classified, according to their chemical composition, into eight *primary* and one *secondary* species. The primary species are: 1. Uric acid. 2. Urates. 3. Oxalate of lime. 4. Cystine. 5. Xanthine. 6. Urostealith. 7. Bone earth (basic phosphate of lime). 8. Carbonate of lime. The secondary concretion is composed of a mixture of the phosphate of lime and the ammoniaco-magnesian phosphate.

In addition to these, which are composed of normal or abnormal, but strictly urinary, ingredients, two other species are occasionally found in the urinary passages which have an origin extraneous to the urine. These are *fibrine or blood concretions* and *prostatic calculi.*

Urinary concretions always contain, in addition to their proper components, slight admixtures of animal matters, viz., mucus, epithelium, pigment, and, generally also, more or less desiccated blood and pus.

The term "gravel" is given to concretions of small dimensions, which are not too large to be spontaneously voided by the urethra; the larger masses are called "stones," or "calculi."

Calculous formations are said to be *primary*, when they are deposited from the unchanged urine, owing to some inherent vice in its composition; and *secondary*, when the deposit is due to ammoniacal decomposition of the urine in the lower urinary passages.

It is essential to recognize this difference in order to understand the mode of growth of urinary calculi, and the principles which should guide their medical treatment.

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[1] The following table shows the proportion of deaths from stone in each of the eleven registration districts of England and Wales, for every 100,000 deaths from all causes, in the ten years 1857–66. Males only are included. (Constructed from the Registrar-General's Reports.)

North-Western	34
South-Western	46
Northern	54
West-Midland	64
South-Midland	71
Yorkshire	75
London	90
Monmouthshire and Wales	91
South-Eastern	93
North-Midland	98
Eastern	115

It has been already explained that whenever the urine becomes decomposed and ammoniacal, its earthy constituents are precipitated as a sediment composed of phosphate of lime and the ammoniaco-magnesian phosphate, often mixed with small quantities of urate of ammonia and carbonate of lime. This is identical with the *secondary* phosphatic deposit on urinary calculi.[1] Its production is due to the transformation of urea into carbonate of ammonia. Any obstacle which delays the urine in its channels, and prevents its speedy and complete evacuation, tends to bring about this change. The presence of a calculus in the bladder presents a condition highly favorable to the production of ammoniacal urine, and to the precipitation of the secondary phosphatic deposit. Accordingly, it is found that calculi which have been long detained in the bladder, are frequently covered over with a phosphatic incrustation. Indeed, it may be said that this is the proper ultimate stage and last chapter in the history of every urinary concretion, unless its career be cut short by spontaneous expulsion, or removal by surgical operation.

The epoch at which the secondary deposit begins to form is quite uncertain, and depends on the concurrence of cystitis. Sometimes small calculi, weighing only a few drachms, are found covered with a thick investment of phosphates; in other instances large calculi, weighing many ounces, are found without any traces of phosphatic incrustation. So long as the urine remains acid the surface of the stone remains free from phosphates, but as soon as the urine becomes freely ammoniacal, the secondary deposit begins to accumulate.

It follows from these facts, that a solvent treatment which may have been applicable in the early existence of a stone, ceases to be so when the urine becomes ammoniacal and a secondary deposit has taken place on its surface.

The principal points relating generally to the structure and growth of urinary calculi are embraced in the following propositions:

1. Calculi may consist entirely of one ingredient, as uric acid, oxalate of lime, cystine, etc.; or two or more primary deposits may alternate with each other in the form of layers, so as to constitute an *alternating* calculus.

2. The most common alternations are uric acid and oxalate of lime; but any primary deposit may alternate with any other primary deposit: as cystine with uric acid; uric acid with bone earth; or oxalate of lime and bone earth. The last two cases, however, are excessively rare. The number of layers composing

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[1] The fetid incrustation which covers public urinals is likewise of similar nature.

an alternating calculus is quite uncertain; there may be only three or four, or twenty or thirty. The thickness of the layers varies conversely with their number.

3. A calculus consisting of only one substance has usually a stratified arrangement, and exhibits an indefinite number of concentric layers. Such is usually the structure of uric acid, oxalate of lime, and phosphatic calculi. But sometimes the calculous matter is deposited in vertical lines radiating from the centre. This is the usual structure of cystine calculi. Sometimes one portion of a stone has a radiated, and another portion a stratified formation.

4. Most urinary calculi are divisible into a central portion or *nucleus*, and an outer portion or *body*. There is also not unfrequently an outer investment, or *crust*, of phosphatic deposit.

5. The nucleus may be of the same nature as the body, or differ from it. The nucleus may consist of uric acid, urates, oxalate of lime, or any other primary formation, or it may be a clot of blood or a mass of mucus; or, lastly, it may consist of some foreign body introduced from without.

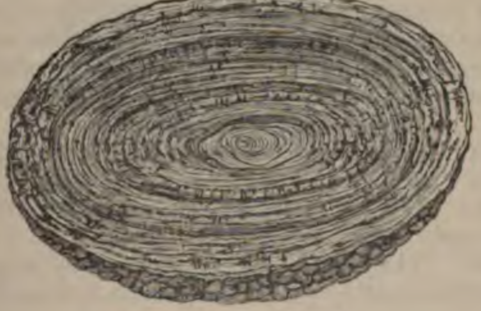
6. The determining *causes* of the formation of urinary calculi are still but imperfectly known. The more usual are the following: (*a*) An excessive proportion of the precipitated ingredient in the urine; (*b*) A too acid state of the urine, which diminishes its solvent power over uric acid and the urates; (*c*) An alkaline state of the urine. If the alkalescence be due to fixed alkali, the bone-earth phosphate and carbonate of lime are liable to precipitation; this is, however, a very rare contingency in the human subject, though common in the herbivora. If the alkalescence be due to carbonate of ammonia, the secondary phosphates are precipitated; (*d*) Deficiency of chloride of sodium and the alkaline phosphates in the urine, reduces its solvent power on uric acid (Heller); (*e*) The presence in the urine of an abnormal constituent of sparing solubility, such as cystine or xanthine; (*f*) The accidental presence of a body suitable to form a nucleus, such as a small mass of concrete blood, mucus, epithelium, or an extraneous body, such as a bit of bougie, a piece of bone, or of a wire or needle, a bit of sealing-wax, and so forth.

Considerable light has been thrown on the *mode of origin* of urinary calculi by an examination of the microscopical structure of their nuclei. Dr. V. Carter found that the actual nucleus consisted nearly always of globular forms of urates and oxalate of lime (dumb-bells and spheroids), and not ordinary crystals of these substances. The researches of Rainey and Ord have shown that these globular forms are only produced when precipitation takes place slowly in a colloid medium; and Carter found that a colloid matrix always exists in the nuclear formations of urinary calculi. It would therefore appear probable that the initial step in the formation of a calculus is the exudation of some colloid—mucus, or some other albuminoid substance—into the urinary passages. Into this colloid, urates or oxalate of lime, or both, are precipitated, and combining with it, form molecular aggregations of a globular character, which constitute the foundation of the subsequent growth. Under what conditions the colloid is exuded cannot be with certainty explained; but the probability is that congestive or subinflammatory states of the kidneys, such as occur in the febrile state, give occasion to such an exudation and supply a starting-point to a process which does not attract attention until after a long lapse of time.[1]

OF THE PARTICULAR VARIETIES OF URINARY GRAVEL AND CALCULI.

1. Uric Acid.—This is by far the most frequent species of urinary concretion. It constitutes probably five-sixths of all renal concretions, and of vesical calculi which have only recently descended from the kidney. As *gravel*, uric acid may be passed in the form of small distorted crystalline agglomerations, or as little smooth spherical bodies, ranging from the size of a poppy seed to that of a mustard seed, or in flattened warty concretions as large as split peas. All these have a yellowish, brownish, or reddish color. They are derived from the kidney, and may be discharged singly or in numbers at irregular intervals.

Fig. 40.



Section of a uric acid calculus.

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[1] *See* Carter "On the Microscopic Structure and Formation of Urinary Calculi." Lond., 1873. Also Dr. Ord's paper, Med.-Chir. Trans., 1875; and his work "On the Influence of Colloids upon Crystalline Form and Cohesion." Lond., 1879.

When retained in the bladder, they grow into flattened oval *calculi*, sometimes roundish, sometimes elongated like an almond. They vary in color from a light fawn to a deep brick-red, according to the quantity and nature of the urinary pigment which they contain. Their surface is usually studded with minute tubercles, or mamillations, which are worn into smooth facets if more than one stone coexist in the bladder. Their weight varies from a drachm to an ounce, but sometimes reaches four or five ounces.

Uric acid calculi possess considerable hardness; their specific gravity is about 1.5. Uric acid is best recognized by the murexid test, previously described. Its most important properties, from a therapeutical point of view, are its solubility in weak solutions of the carbonates of lithia, potash, and soda, and its insolubility in strong solutions of the bicarbonates of potash and soda, as well as in water and dilute acids.

Pathologically, uric acid is closely related to gout. Hence, the frequency of uric acid gravel and stone in the wealthier classes in the middle and later periods of life.

The urine, in the subjects of uric acid calculi, is acid, and often high-colored, prone to deposits of uric acid crystals and amorphous urates,

The medical treatment of this class of calculi will be described at length in a separate section. (*See* Solvent Treatment.)

2. Urate Concretions.—The same confusion has existed respecting the composition of these concretions, as respecting that of the amorphous urate deposit. They are usually designated urate of ammonia, but their chemical nature requires reëxamination.

They constitute small, soft agglomerations in the kidneys—rarely in the bladder; and are almost confined to young children. Heller[1] states that he has found them several times in the kidneys and ureters of sucking infants in the Vienna Foundling Hospital. They formed small irregular clumps, sometimes heaped together into a mass as large as a kidney bean. Heller encountered similar calculi on two occasions in adults. Calculi wholly composed of urates are very rare, and never reach a large size; but globular masses of urates nearly always exist in the centre of a urinary calculus, although its bulk may be formed of some other ingredient.

The deposition of clumps of urate of soda in the urinary passages is not uncommon in the febrile complaints of infants and young children; and it seems not unlikely that some of these clumps may be retained in the pelvis of the kidney or in the bladder, and become the nuclei of future calculi; and that the excessive frequency of calculi in children is due to this cause (*see* p. 98).

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[1] Harnconcretionen, p. 134.

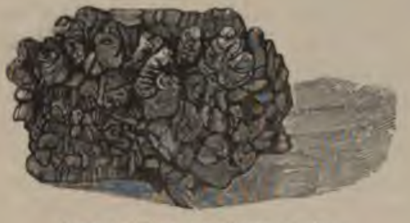
The urine from which this variety of concretion is deposited, has an acid reaction, and the medical treatment is identical with that of uric acid calculi. The circumstances under which this concretion is deposited, must be carefully distinguished from those in which urate of ammonia (of undoubted composition) is deposited in an ammoniacal urine mixed with secondary phosphates.

Urate concretions are distinguished chemically by their solubility in hot water.

3. OXALATE OF LIME OR MULBERRY CALCULUS.—Oxalate of lime may be discharged as minute concretions, or gravel, from the kidney, or grow to be a stone in the bladder. In the former case the concretions are usually smooth, rounded, grayish-dark bodies, resembling hemp-seed.

Vesical calculi of this class are exceedingly hard, and break into sharp angular fragments when crushed by the lithotrite They are usually of a spherical shape; their surface is tuberculated like a mulberry (Fig. 41), and is usually of a blackish-brown color. Sometimes, however, they are oval and smooth, and of a bluish-gray color.

FIG. 41.



Oxalate of lime or mulberry calculus.

The nucleus of a mulberry calculus is frequently composed of uric acid; and, conversely (though much more rarely), a uric acid stone may have a nucleus of oxalate of lime. Beale and Carter have further shown, that in the centre of a uric acid nucleus, there is often a microscopic clump of dumb-bells of oxalate of lime.

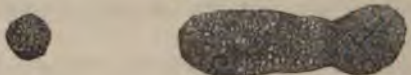
Calculi composed of alternate layers of oxalate of lime and uric acid are more common than those composed of oxalate of lime alone. These layers may form complete concentric capsules, or be partial and imperfect. In the latter case the concretion is amenable to the solvent and disintegrating action of the alkaline carbonates; in the former it is wholly beyond the power of such solvents.

Oxalate of lime is insoluble in alkaline carbonates and organic acids; but it dissolves in nitric and muriatic acids. When heated before the blowpipe, it first blackens, and finally leaves a bulky white ash of caustic lime, which blues moistened litmus paper.

During the formation of oxalate of lime calculi, the urine is always acid.

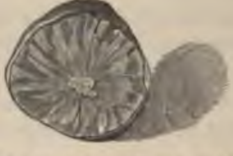
4. CYSTINE.—Gravel and calculi of cystine belong to the rarer species of urinary concretions. They are usually found in the bladder as large calculi, but sometimes they are discharged spontaneously as gravel. I have in my collection two examples of pure cystine calculi passed spontaneously (*see* Fig. 42). One of them is a small lenticular mass weighing a grain and a half. The other is cylindrical in shape, an inch and a quarter in length, and weighing twenty-seven grains. Both have a crystalline granular surface and a light yellow color. Sometimes vesical calculi of cystine attain a weight of three or four ounces. They are usually egg-shaped, of a full honey-yellow color, mamillated on the surface, and lustrous, as if studded with minute crystals. When cut into, they show a radiated structure, and an obscurely transparent brilliance like yellow beeswax. They are usually composed of pure cystine, unmixed with any other substance. Sometimes they have a nucleus of uric acid. In a specimen in the Museum of Owens College (Fig. 43) the central nodule is uric acid; around this is a body of pure cystine; overlying this a layer of mixed uric acid and cystine; and enveloping the whole a crust of secondary phosphates, mixed with cystine.

FIG. 42.



Cystine calculi spontaneously voided.

FIG. 43.



Section of a cystine calculus, with a nucleus of uric acid, and an external coat of phosphate.

Cystine calculi possess the curious property of assuming a pale green color when long exposed to full daylight. The specimen just referred to afforded an interesting example of this change. The calculus had been divided equatorially; one-half lay in the cabinet with its cut surface downwards, and the other half with the cut surface upwards, exposed to the light. The latter had a delicate emerald-green tint, while the former preserved its original yellow color.

Another curious circumstance in the history of cystine, is its tendency to run in families. Dr. Marcet gives an account of two brothers in whose kidneys cystine calculi were found. Both Lenoir and Civiale extracted cystine calculi from the bladders of two brothers. Toel relates the history of two sisters and a mother who voided cystine with the urine. Ebstein[1] also has described cystinuria as occurring in two brothers.

Cystine calculi are much more friable than uric acid or oxalate of lime. They are easily scraped with the nail, and offer especially favorable objects for treatment by lithotrity. My late colleague, Mr. Southam, showed me a quantity weighing ninety grains of the fragments of a pure cystine calculus, which had been voided by a little girl four years of age after a single crushing.

Cystine is recognized with great facility. If a particle be placed on a watch-glass, or on a slip of glass, and treated with caustic ammonia, it speedily dissolves; by exposure to the air for a few hours, the volatile alkali exhales, and beautiful six-sided crystals are deposited, which are highly characteristic (*see* Figs. 14 and 15). Cystine is also soluble in the mineral acids; and in the fixed alkalies and their carbonates; but it is precipitated by organic acids and by carbonate of ammonia.

5. XANTHINE calculi are excessively rare. (*See* Xanthine, p. 111.)

6. FATTY OR SAPONACEOUS CONCRETIONS. UROSTEALITH (of Heller).—In the Museum of the College of Surgeons of London there are two magnificent specimens of vesical calculi, composed of a central fatty or saponaceous mass surrounded with a thick investment of phosphates (Fig. 44). Both belonged to Hunter's collection, and both are figured and described in the catalogue of calculi published in 1842. They are described as "consisting of the earthy phosphates deposited upon a mass of oleate and margarate of lime." This mass is of a light yellow color, and its irregularities correspond with those of the cavity in which it loosely lies. At p. 129 of the catalogue, the following ingenious remarks are made respecting the probable origin of these stones: "On account of some real or supposed disease of the bladder, a solution of soap has been injected into

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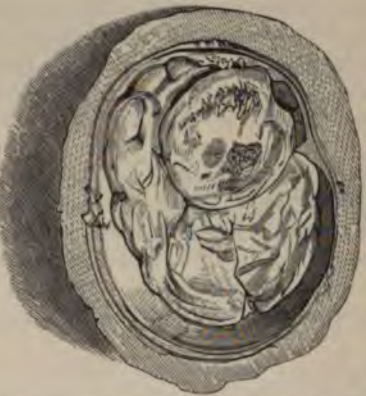
[1] Deutsch. Arch., Bd. xxiii.

its cavity; mutual decomposition between the soap and the salts of the urine has been the necessary result; the alkali of the former uniting with, and forming soluble compounds with, the phosphoric and other acids of the urine, while the earthy bases of the urine have precipitated, in combination with the fatty acids of the soap, in the form of a semi-gelatinous sparingly soluble compound, being in fact an earthy soap; this substance, acting as a foreign body in the bladder, has induced the deposition of the phosphates, and given rise to the formation of a calculus."

FIG. 44.



Section of a fatty or saponaceous concretion (*urostealith*) surrounded with phosphates. From the Museum of the London College of Surgeons.

The fatty or saponaceous masses here described are probably of the same nature as those described by Heller in 1845, and named by him *Urostealith.*[1] Only one other case has been published; it was observed by Dr. W. Moore in 1853.[2]

Heller's patient was a man, 24 years of age, who passed a number of small concretions about as large as peas. When fresh, they were soft and elastic, like India-rubber, but dried into hard, brittle, wax-like masses. They dissolved readily in caustic potash, forming a soap. They also dissolved readily in ether, but with difficulty in alcohol. In hot water they did not dissolve, but softened. They melted with heat, and eventually burned with a bright yellow flame, exhaling an odor of shellac and benzoin. They contained a large quantity of earthy phosphates.

Dr. Moore's specimens consisted of two very small dark brown calculi, which had a soft wax-like consistence, and appeared to consist of a lime soap. They partly dissolved in hot alcohol; and the solution, when cold, deposited a whitish matter, which exhibited numerous fat globules, but no crystalline plates. When incinerated before the blowpipe they yielded a white, alkaline, calcareous ash. A year later, Dr. Moore received from Dr. R. Adams two calculi taken from the body of this patient. One was a large phosphatic stone, in the centre of which was a cavity containing some of the same dark brown substance. Dr. W. Davy, who examined a portion of this, judged it to be composed of lime "in combination with the fat or waxy substance forming some organic combinations with the fatty acids."

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[1] Harnconcretionen, p. 146; also Heller's Archiv, Bd. ii. p. 1.

[2] Dublin Quarterly Journ. of Med. Science, vol. xvii. p. 478.

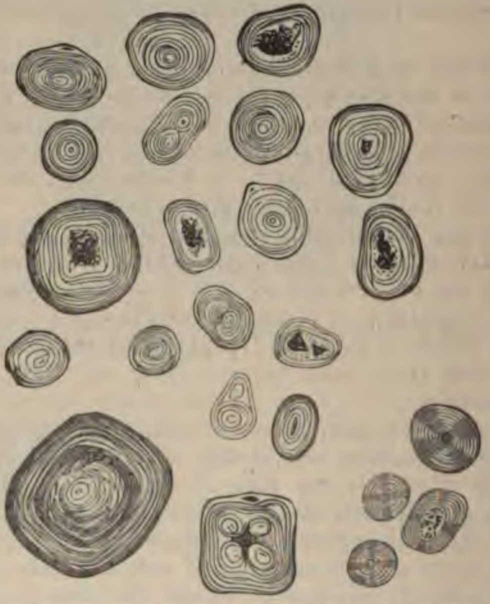
7. Carbonate of Lime.—Concretions of carbonate of lime are very rare in the human subject. Those which have hitherto been encountered were of small dimensions, varying from the size of the smallest visible granules to that of a hazelnut, smooth on the surface, gray, yellowish, or bronze colored—sometimes with a metallic lustre, and generally very hard.

The following remarkable case, in which myriads of minute calculi of carbonate of lime were voided with the urine, was described in the first edition of this work as an example of the spontaneous expulsion of prostatic calculi, but a communication I have since received from Dr. Haldane, of Edinburgh, has convinced me that they were derived from the kidneys, and not from the prostate.

The patient was a gentleman seventy years of age, suffering from enlarged prostate, under the care of Mr. George Hunstone, of this city. On the 20th of April, 1864, Mr. Hunstone brought me a specimen of the urine for examination. It was ammoniacal, and contained a good deal of pus. At the bottom of the phial were a large number of minute amber-colored calculi—the largest of which were about the size of poppy seeds, and the smallest only just visible to the naked eye, as bright specks. On subsequent occasions Mr. Hunstone brought me additional quantities of urine containing similar bodies. Altogether I obtained about eight grains of these calculi; they were easily separated from the urine by levigation and decantation. Mr. Hunstone stated that the patient had been in the habit of voiding these calculi for some months, at frequent intervals. The patient died some few months afterwards, and, unfortunately, no opportunity was afforded of making a post-mortem examination.

The largest of the specimens in my possession is about the size of a mustard-seed; there are a good many as large as poppy seeds; but several hundreds are less than a quarter of this size, and many thousands are still smaller. They are mostly spherical in shape; many are rudely cubical or pyramidal. They possess a full amber color, and are finely translucent. Under the microscope they present the appearance represented in Fig. 45, and exhibit an infinite series of concentric lines. The centre or nucleus is variously composed. In some of them it is an object resembling a glandular cell, in others a prismatic crystal, in others amorphous earthy-looking matter. In some, again, the nucleus is double; in others, treble, or even quadruple (Fig. 45). With polarized light they display a dark cross, as represented in the lower right-hand corner of the figure. When crushed they break into angular fragments.

Fig. 45.



Carbonate of lime calculi spontaneously voided with the urine—highly magnified.

The calculi dissolve rapidly in mineral acids, with abundant disengagement of carbonic acid—leaving ragged, brown, flaky, organic remnants. Acetic acid acts very slowly upon them, without visible disengagement of carbonic acid; but in the course of two or three days all the mineral matter is taken up, and the animal matrix is left, as soft, light balls, preserving the stratified appearance of the original calculi, but with a diminution of their original translucency. They are unaffected by caustic potash. The murexid test yields not the slightest evidence of uric acid. When heated to whiteness before the blowpipe, their surfaces fuse into a brilliant iron-gray enamel, which protects the deeper parts. As the incandescent calculi cool, the enamelled surfaces crack into numerous minute polygonal spaces. The solution of the calculi in muriatic acid throws down an abundant, white, flocculent precipitate when saturated with caustic ammonia. These reactions indicate that they are composed of an animal matrix impregnated with carbonate of lime mixed with a little phosphate of lime.

Dr. Haldane's communication above referred to contains an account of a case in which calculi, identical in every respect with those just described, were found in the kidney after death. Dr. Haldane's notes are as follows:

"W. A., a mason, aged 33, was admitted into the Edinburgh Infirmary under Dr. Gillespie, on the 15th of October, 1856. Fifteen months before admission he strained his back while engaged in raising a large stone.

"Two months afterwards an abscess formed over the upper part of the sacrum, which was poulticed, opened, and matter was discharged.

"The abscess healed up, but matter again formed; the second abscess burst about two months before his admission into hospital. During the greater part of the fifteen months he had been at his work.

"When admitted into the hospital he complained of pain in the back and general weakness. There was an opening at the right side of the sacrum, from which pus discharged. A probe could be introduced its whole length, and seemed to pass towards the anterior part of the lumbar vertebræ. He sank gradually, and died on the 21st of April, 1857.

"No urinary symptoms were noticed during his residence in the hospital.

"I examined the body on the 22d of April. A large accumulation of matter was found in front of the bodies of the lumbar vertebræ; there was caries of the anterior part of the bodies of the first four. The spinal cord was not affected.

"The heart and lungs were natural.

"The liver was small, weighing 31 ounces, but was healthy in structure.

"The right kidney weighed 5 ounces; it was anæmic but healthy, except that a few gritty particles were embedded in some of the cones. These were found to consist partly of carbonate of lime.

"The left kidney weighed 4½ ounces. In the pelvis was about half a teaspoonful of sandy-looking material, held together by a flocculent substance, which resembled coagulated blood. The sandy matter was in small particles, generally about the size of grains of sand; some were as large as hemp-seeds. The lining membrane of the pelvis was thickened, and at some points appeared slightly abraded. The kidney was a little atrophied, owing to dilatation of the pelvis. The ureters were natural. The bladder was contracted; it contained about a teaspoonful of urine, which unfortunately was not examined.

"I looked upon this case as possibly an example of the calcareous metastasis described by Virchow. When in connection with absorption of bone, carbonate and phosphate of lime are deposited elsewhere."

Dr. Haldane was good enough to send me a sample of the calculi obtained from the kidney in this case; and I had no difficulty in deciding on their absolute identity both in naked-eye and microscopic appearance, as well as in chemical composition, with those examined by myself. It seems also highly probable that the history of their occurrence was strictly analogous.

In the nineteenth volume of the "Transactions of the Pathological Society," Mr. Wagstaffe describes a "large branching calculus," composed of carbonate of lime, removed after death from the right kidney of a man aged forty-two. Several small ones, of similar composition, were also found in the same kidney. Dr. Ord also has described calculi composed of carbonate of lime.[1]

8. Basic Phosphate of Lime or Bone-earth.—Concretions of this substance alone are very rare. They were formerly confounded with the mixed phosphates which constitute the secondary deposit. They vary in size from a pea to a hen's egg. They are white and chalky in appearance, and of a soft, smoothish exterior, with an earthy fracture. Sometimes their texture is loose, sometimes very compact.

Bone-earth rarely alternates with any other deposit; occasionally, however, it does so. There is a fine specimen in the museum of the Manchester Infirmary, in which bone-earth alternates with uric acid.

When the urine is rendered alkaline by alkalizing salts, or becomes alkaline after a meal, the bone-earth phosphate is sometimes abundantly deposited; but, from its uncrystalline condition, it has very little tendency to agglomerate into concretions. Patients may pass an alkaline and turbid urine (from this cause) for months, without practically any risk of the formation of a stone.

9. Mixed or Secondary Phosphates (Fusible Calculus).—The composition and production of this deposit from ammoniacal urine has been already explained. It rarely forms the centre of a urinary calculus; but more commonly encrusts calculi of some other species, or an extraneous body which acts as a nucleus (Fig. 46). Concretions of this substance are frequently formed around the inequalities of fungous or other growths of the urinary organs. Calculi of the mixed phosphates may go on increasing for an indefinite period, and completely fill the bladder, attaining a weight of 10, 20, or even 30 ounces.[2]

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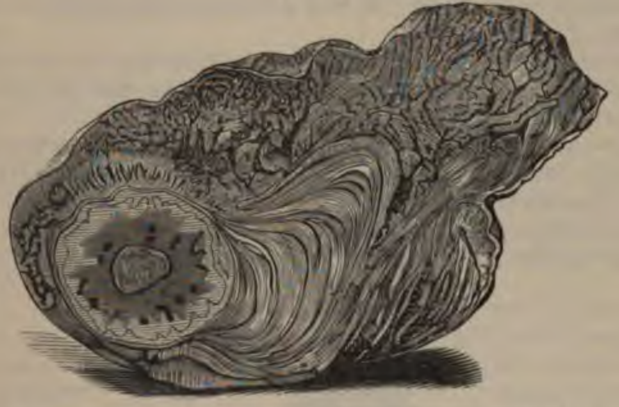
[1] "On the Influence of Colloids," etc., p. 135.

[2] Dr. Utterhoeven, of Brussels, withdrew, by the suprapubic operation, from the bladder of a man, aged thirty-nine, an enormous oval concretion weighing 40½ ounces, and measuring round its longest diameter 17 inches. I believe this is the largest ever extracted from a living person. It had been growing from the age of twelve. (Leroy d'Etiolles (fils), Traité pratique de la Gravelle. Paris, 1863.)

In their physical characters, fusible calculi most resemble the bone-earth phosphate. They are usually lax and friable, composed of concentric laminæ, or irregular; often studded on the surface with brilliant glistening points of triple phosphate crystals. They readily break down under the lithotrite; but the general irritation of the system, and the frequent coexistence of grave anatomical lesions in the urinary passages or the kidneys, render these cases unfavorable subjects for operation. They are especially suited for a solvent treatment by means of acid injections, thrown into the bladder in the manner recommended by Sir B. Brodie.

FIG. 46.



Section of a concretion, consisting of a vast mass of the mixed phosphates deposited on a calculus of oxalate of lime.[1]

Chemically, this concretion is characterized by fusing into an enamel when strongly urged by the blowpipe. It is very soluble in acids, especially the mineral acids; but wholly insoluble in water and alkalies.

10. FIBRINE AND BLOOD CONCRETIONS.—Marcet gives an account of a small calculus about the size of a large pea, which was passed, after much suffering, by a gentleman between 50 and 55 years of age. He had been suffering for two or three years from symptoms of urinary calculi, and had previously passed three similar concretions. The specimen examined by Marcet had a yellowish-brown color, somewhat resembling beeswax. Its hardness was also nearly that of beeswax. Its surface was uneven, but not rough to the touch; it was somewhat elas-

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[1] From a drawing in the possession of Mr. Southam. The history of this stone (which was successfully removed by the recto-vesical operation) is given by Mr. Southam in the 42d volume of the Medico-Chirurgical Transactions.

tic. When examined chemically, it answered to the reactions of fibrine.

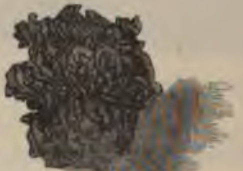
A concretion, about the size of a small pea, was handed to me for examination by my late colleague, Mr. Beever. It had been passed by a man of thirty-five, whose urine was not albuminous. Its texture was hard and brittle, its external surface rough, its color dark reddish-brown. It swelled into a voluminous coal under the blowpipe, and, when fully incinerated, left a very scanty white ash. It was evidently composed of inspissated blood.

A patient whom I saw with Dr. Holland was in the habit of passing numbers of blood concretions of a softer texture. He had previously suffered from hæmaturia.

Numerous similar concretions were found loose in the infundibula and pelvis of the kidney, in the case of ruptured kidney already described at p. 153.

My collection contains a very fine blood concretion, taken from the bladder of a sheep (Fig. 47). The specimen was presented to me by Mr. Lund. It is as large as a small walnut, very light—weighing only 37 grains. It is nearly spherical, and exceedingly rugged on the surface, which is studded all over with reddish-black warty projections. This dark warty part forms the outer crust of the concretion, is very brittle, and breaks with a lustrous fracture. When sawed through, the rough outside crust is found to be about a line thick: it invests an oval body, which has an even, sharply defined outline. The body has the appearance of baked clay; it is of nut-brown color, and easily scraped with the nail. It breaks with a dull fracture, like a piece of catechu. Examined chemically and microscopically, both body and crust were found to possess the characters of concrete blood. The scanty ash obtained by calcination gave abundant evidence of iron.

FIG. 47.



Blood concretion from the bladder of a sheep.

All these instances appear to have been connected with the occurrence of renal hæmaturia. Such concretions sometimes serve as nulclei for uric acid or oxalate of lime calculi.[1]

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[1] Wilson's Lectures on the Urinary Organs, p. 81.

11. Indigo.—Only one example of calculus composed solely of indigo is known. It was described by Dr. Ord in the "Pathological Transactions," vol. xxix., and was found in the pelvis of the right kidney of a woman, who had died from a sarcomatous tumor of the left kidney. The calculus was flat and hard, and weighed 40 grains. Its color was dark brown in some parts, in others blue-black, and it left a blue-black mark when drawn across a sheet of white paper. On heating it gave off the odors of burnt indigo, and sublimed in blue prisms of indigo.

12. Prostatic Calculi.—Although these are not, strictly speaking, urinary products, they are in very rare instances discharged spontaneously with the urine, and therefore deserve some notice in this connection.

Sir H. Thompson, who has investigated this subject with great care, states that the existence of concretions in the prostate is almost universal after the age of puberty. He found them invariably present in seventy prostates which he examined from persons above twenty.[1]

They begin as minute, globular, transparent bodies in the follicles of the gland. At first, they are wholly composed of an albuminous matter, arranged in concentric layers around a vesicular nucleus. But as they grow, they are gradually more and more impregnated with mineral matter, until at length they attain the hardness of the hardest urinary calculi. As a rule, they produce no symptoms, and their existence is, perhaps, hardly to be looked on as a disease. They usually vary in size from a poppy seed to $\frac{1}{2000}$ of an inch.

In some cases, however, the process does not stop here. The earthy material is deposited in great quantity; and large concretions are formed, which encroach on the glandular tissue, and project into the urethra in the form of oblong masses, which require operative procedures for their removal.

ON THE DIAGNOSIS OF THE SPECIES OF URINARY CALCULI WITHIN THE BLADDER OR KIDNEY.

It would greatly facilitate the choice of the most appropriate treatment, in an individual case of urinary calculus, if it were possible to ascertain beforehand the exact nature of the concretion. This remark applies equally to surgical and to medical treatment, but more strongly to the latter than to the former.

The degree of precision of this knowledge attainable in different cases varies a good deal. The most certain knowledge is gained when a person who has been in the habit of spontaneously voiding small concretions becomes afterwards the subject of stone. In such a case, the examination of the calculi previously passed (supposing them to have been preserved) throws a sure light on the nature of the one retained—provided the epoch at which the former were voided be not too remote, and the characters of the urine continue to correspond.

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[1] Sir H. Thompson, On the Enlarged Prostate.

In the absence of this kind of evidence, certain knowledge of the nature of the stone is often unattainable; but still, it is generally possible to indicate—from the character of the urine, the microscopic examination of the urinary deposit, the constitution of the patient, and the known relative frequency of the several species of stone—*with strong probability*, the species to which it belongs; and also (and with still greater certainty) some of the species to which it does *not* belong.[1]

With regard to the character of the urine, the most important indications are supplied by the nature of its reaction, and the character of the deposit which may be precipitated from it. The reaction of the urine may be (*a*) acid; (*b*) alkaline from fixed alkali; or (*c*) alkaline from carbonate of ammonia.

a. If the urine be *acid*, the stone is almost sure to be uric acid or oxalate of lime, or a mixture of these two.[2] These two deposits alternate with each other so frequently, and at such short intervals, that, if the urine be free from a uric acid or an oxalate of lime sediment, there is nothing to indicate directly which of the two species the stone belongs to. But, as uric acid is much more common than oxalate of lime, the probabilities are considerably in favor of the former. If the urine, on cooling, deposits abundantly either uric acid or oxalate of lime, and *à fortiori*, if either of these deposits is found in the urine at the moment of emission, there is strong probability that the *surface* of the stone is of the same nature; but this gives no warrant of the composition of the deeper strata.

Vesical calculi are usually more complex in their composition than renal calculi. The latter are almost always composed of one single ingredient; but the former are frequently composed of more than one ingredient. The longer a calculus has resided in the bladder, the more complex will its composition probably be; and conversely, the more recent its descent from the kidney, the more likelihood that it is composed of but a single ingredient. If, therefore, the urine be acid, and the calculus of recent date, the probabilities are greatly increased that it is composed of uric acid alone.

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[1] For the diagnosis of renal calculi and of their chemical constitution, *see* Chapter VII.

[2] Concretions of xanthine, fibrine, and fatty matters are altogether left out of consideration, on account of their extreme rarity. Cystine is also excessively rare; and if cystine crystals be not found in the deposit, it may, practically, be likewise excluded.

As mulberry calculi have rough surfaces, they usually produce more violent irritation of the bladder than the smoother stones composed of uric acid. This indication is, however, of little practical value, and the exceptions to it are numerous.

Persons of gouty disposition are more likely to be subjects of uric acid than of oxalate of lime calculi.

b. If the urine be *alkaline from fixed alkali*, the stone will be composed either of bone-earth phosphate or carbonate of lime. Both are of extreme rarity.

c. If the urine be *alkaline from carbonate of ammonia*, the composition of the nucleus and body of the calculus can no longer be divined; but its surface or crust is sure to be composed of the mixed phosphates. The depth of this crust can only be conjectured from the intensity of the ammoniacal reaction, the quantity of pus and flakes which are discharged with the urine, and the length of time during which this state of urine has persisted. Care must be taken to ascertain if the urine be ammoniacal *at the moment of emission;* for in most cases of stone there is some degree of cystitis; and the presence of pus causes a urine which was passed acid, speedily to become ammoniacal. The degree of the ammoniacal reaction is best judged of by the intensity of the ammoniacal odor, by the gelatinized, or loose, condition of the pus, and by the abundance of triple phosphate crystals. If the urine be only very feebly ammoniacal, or have only recently become ammoniacal, the phosphatic crust may be only a thin film. In the case of large or old phosphatic concretions, fragments of phosphatic *débris* are frequently voided with the urine. If the ammoniacal reaction of the urine is once established in a case of stone, it seldom afterwards gives place to an acid reaction.

MEDICAL TREATMENT OF GRAVEL AND CALCULI.

Two objects are to be held in view in the medical treatment of gravel and calculi, namely (A), to prevent the formation of a concretion when a tendency thereto exists; and (B) to dissolve or facilitate the expulsion of concretions already formed. The treatment of the organic lesions which are incidental to the presence of calculi in the urinary passages will be considered—in so far as they implicate the kidneys and their immediate appendages—in Part III. with the other organic affections of the kidneys.

(A) Preventive Treatment.

The disposition to the production of gravel and stone generally passes by undetected, until a concretion is actually formed. The general health is, usually, not markedly disturbed, and the local symptons only attract attention when the urinary passages begin to resent the presence of the foreign body.

Sometimes, however, the practitioner becomes aware beforehand, from the character of the urine or other circumstances, that the formation of a stone is a probable event unless preventive means be adopted.

The occurrence of a deposit of uric acid or oxalate of lime, after the urine has stood some hours, indicates no special risk of the formation of a stone; but if either of these substances be voided *with* the urine, as gravel, such a risk does certainly exist, and demands to be provided for. Again, if the urine, although clear when voided, lets fall a crystalline deposit *before it has completely cooled*, as may sometimes be seen, especially in children, the danger of a formation of stone should not be overlooked. The presence of cystine in the urine is, at all times, a circumstance which demands precautions against the formation of a calculus. The existence of an ammoniacal state of the urine, also, always involves a risk of the deposition of the secondary phosphates.

Independently of the existing state of the urine, evidence of a calculous tendency is sometimes obtained from the antecedents of the patient. If the patient have recently voided a concretion with the urine, or if one have been removed from his bladder by surgical operation, there is reason to apprehend a continuance of the calculous tendency, and the formation of a new concretion.

Under any of these circumstances, preventive measures are demanded. These may be divided into *general* and *special*. The former apply to calculous tendencies of every kind; the latter to threatened formation of some particular species of stone.

Among the *general* indications, the most important is to obviate undue concentration of the urine. This is effected by the systematic use of increased quantities of aqueous drinks. The urine is apt to reach the greatest degree of concentration at hours remote from meal-times (especially during the two or three hours which precede a late dinner), and during the hours of sleep. At these periods the flow of the urine is exceedingly scanty; it is long delayed in the bladder before there is any call for its evacuation; its solid constituents are in excessive proportion to the watery parts, so that the urine resembles a supersaturated saline solution; it is also very acid. Here are united all the conditions most favorable to the separation of some of its less soluble components. Dr. Prout pointed out that the recumbent posture during sleep furnished an additional source of apprehension, inasmuch as the urine is no longer aided in its descent by the force of gravity; it therefore lingers and accumulates in the pelvis of the kidney, and is liable to deposit some of its constituents therein.

All these untoward contingencies are obviated by the simple expedient of taking a tumbler of water a couple of hours before dinner, and another before retiring to rest. By this means the urine is diluted, and its escape hastened at the periods when it would otherwise be dangerously saturated, and unduly delayed in the excretory conduits. Two other points are worthy of attention, with a view of maintaining the urine in a state of safe dilution, and providing for its undelayed expulsion. These are: first, that a too great interval shall not elapse between any two meals; and, secondly, that the period devoted to rest in bed shall not be too prolonged. From observations recorded in a previous page, it is seen that a meal both renders the urine more abundant, and lessens its acidity. An individual who shows a tendency to calculous formations should, therefore, be directed to take four or five light meals during the day, at about equal intervals, and to arise betimes in the morning.

When the nature of the calculous tendency is ascertained, either from the character of the deposit, or from the composition of a previously voided concretion, further and *special* precautions should be recommended.

If the tendency be to the precipitation of *uric acid*, the acidity of the urine should be lowered by the moderate employment of the bicarbonate or citrate of potash. A drachm of either salt may be taken in a tumbler of water at bed-time, and again on rising in the morning. The diet should be regulated in such manner that animal flesh shall not form a too prominent part of it. Rich wines and heavy meals must be strictly prohibited, and a bland, mostly farinaceous, diet substituted.

The deleterious effects of high diet on uric acid gravel, is aptly illustrated in an example furnished by Magendie:

> Mr. ——, a merchant in one of the Hanseatic cities, possessed in 1814 an ample fortune, and he lived in accordance with his means—kept a good table, and indulged in its pleasures freely. He was at this time tormented with gout and gravel. Unexpectedly, he lost all his fortune through a political crisis, and was obliged to take refuge in England, where he lived more than a year, almost in poverty, amid numerous privations; but his gravel completely disappeared. Little by little he succeeded in repairing his affairs; he resumed his old mode of life, and the gravel was not long in reappearing. A second reverse robbed him in a short time of all he had gained. He passed into France almost without resources, and his regimen was consonant to his means; the gravel disappeared. Once again his industry restored him to a life of plenty and ease; he abandoned himself again to the indulgences of the table, and with them appeared once more his old enemy the gravel.[1]

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[1] Magendie, De la Gravelle, p. 25.

The special preventive treatment of *cystine* concretions is identical with that of uric acid.

With regard to *oxalate of lime*, the principal indications are, to dilute the urine by abundant regulated potation of aqueous drinks, and to encourage the action of the skin by baths, frictions, the use of flannel clothing, and exercise in the open air. It is important also to guard against the use of certain vegetables which contain large quantities of oxalates and superoxalates in their tissues. The general use of rhubarb tarts in this country in the spring months, and the use of sorrel as salad in France, are probably frequent causes of oxalate of lime concretions. Magendie records two cases in which it appeared highly probable that mulberry calculi had been produced by the daily use of sorrel (loc. cit. p. 121). Both these articles should be absolutely forbidden.

Mineral and potable waters, which are rich in lime should likewise be avoided.

Heller recommends alkaline substances, on the grounds that oxalate of lime long digested with alkaline carbonates is resolved into a soluble oxalate, and that uric acid is the source of the oxalic acid which appears in the urine. Experiments performed by myself on mulberry calculi, yielded no evidence that the alkaline carbonates exert any solvent action thereon. As to the second point, alkalies do *not* prevent undue formation of uric acid, but merely facilitate its elimination. Nevertheless, I have seen instances in which rendering the urine freely alkaline, caused an oxalate of lime deposit to disappear temporarily from the urine.

Basic phosphate of lime and *carbonate of lime* (unmixed with triple phosphate) are among the rarest forms of urinary calculi. Against them, the proper precautionary measures are, to endeavor to remove the alkalescence of the urine by the exhibition of carbonic acid water, and to exclude as much as possible all articles of food and drink which are rich in calcareous salts.

The precipitation of the *secondary phosphates* frequently requires precautionary measures to prevent calculous concretions. If severe cystitis follow lithotomy or lithotrity, there is cause to fear a deposition of phosphatic matter upon some fragment left in the bladder, or on a mass of inspissated pus and mucus. Indeed, whenever the urine is highly ammoniacal, the same danger is not remote. To guard against it, the irritation of the bladder should be allayed by appropriate means, and the viscus should be thoroughly washed out, at least twice a week, with water, or with a solution containing a drachm of the commercial muriatic acid to a pint of water.

(B) SOLVENT TREATMENT.

For therapeutical purposes, urinary calculi may be divided into two classes, viz., those which are *soluble in alkalies*, and those which are *soluble in acids*. To the former category belong uric acid, the urates, and cystine; to the latter, phosphatic and mulberry calculi. Those which are soluble in alkalies may (conceivably) be attacked by alkalizing the urine by means of certain salts administered by the mouth, or by injecting alkaline solutions into the bladder. Those which are soluble in acids can only be attacked by the latter method, inasmuch as acids cannot be made to pass through the kidneys, save in insignificant proportions.[1]

It will, however, be shown in the sequel, that alkaline substances used in the way of injections, act so feebly on uric acid calculi, that no useful results can be expected from their operation; also that mulberry calculi are unassailable by any solvent method hitherto proposed; so that the solvent treatment of urinary calculi resolves itself, practically, into two lines of action, viz., attacking uric acid calculi (and their congeners), by alkalizing the urine by means of medicines administered internally, and phosphatic calculi, by injecting acid solutions into the bladder.

It is a noteworthy fact, that alkaline substances had obtained an extended reputation in the treatment of calculous disorders, long before the composition of urinary calculi had been discovered. In 1739, a remedy of this class—the nostrum of Joanna Stephens—made so great a noise, that Parliament appointed a commission of professional men to inquire into its virtues. The commission reported favorably, and a reward of £5000 was assigned to Miss Stephens for the secret of its composition. The active ingredients in this nostrum were burnt egg-shells and snails, with Alicant soap. As soon as the secret was divulged, soap, soap-ley (solution of caustic potash), and lime-water, were tried in all kinds of calculous cases. The indiscriminate use of the remedies led, as might have been anticipated, to contradictory results. Both successes and failures were published in large numbers;[2] and opinion was much divided as to their utility. About this time the successes of Cheselden gave a great impulse to lithotomy, and the use of solvents gradually fell into discredit. The subject was resuscitated in France about a century later, under the inspiration of the great advances then made in chemical science, and especially of the discoveries of Wollaston and Fourcroy into the nature and composition of urinary calculi. The virtues of the alkaline bicarbonates—and more particularly of the bicarbonate of soda, the active ingredient of the Vichy springs—were brought into prominence; and a considerable number of cases, successfully treated by these means, were published by Chevallier and Ch. Petit. But again, the absurd claim of universal efficacy brought the solvent treatment into contempt; and for the last twenty years, or more, urinary calculi have been almost wholly abandoned to the surgeon.

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[1] Various attempts have also been made to apply galvanism to the solution of stones in the bladder. It was ingeniously conceived that the decomposition of a solution of nitrate of potash within the bladder, by a galvanic current, would set free, simultaneously, both nitric acid and caustic potash—one or other of which is capable of acting on every variety of stone. But the mechanical difficulties of this proceeding have hitherto proved insurmountable; and the slow action which my experiments prove solutions of caustic potash to have on uric acid calculi, and solutions of uric acid on mulberry calculi, render it hopeless ever to obtain results of practical utility by this method. *See*, also, Heller's Harnconcretionen, p. 99.

[2] Ploucquet gives a list of more than forty papers and pamphlets, published on the subject about 1740.

A perusal of the literature of these two periods, however, strongly suggested the desirability of subjecting the question to a new examination, with a view of ascertaining the causes of the discrepant experience of past times; and also of indicating with some approach to certainty, what may be rationally expected from a solvent treatment, in what cases it is applicable, and the precise mode of carrying it out effectually.

For the purpose of clearing up these questions, the present writer undertook an extensive series of experiments, and made numerous clinical observations. The facts observed are embodied in a paper read before the Medical and Chirurgical Society, on March 28, 1865. To this paper the reader is referred for fuller details. The results obtained seem to demand a considerable modification of the prevailing opinion regarding the inutility of the solvent treatment. They do not by any means indicate the *general* possibility of substituting a solvent for a mechanical treatment of *vesical* calculi; but they suggest an essential improvement in the treatment of *renal* calculi; they also indicate that uric acid calculi, under certain circumstances, are capable of solution in the bladder, by means of alkaline salts administered by the mouth, at a rate which admits of practical application; and that, in certain picked cases of this class, a solvent treatment deserves to be resolutely tried, before having recourse to the more dangerous methods of lithotomy and lithotrity.

Attention was naturally directed in the first instance, and chiefly, to uric acid, both on account of its being by far the most common constituent of urinary calculi, and also on account of its offering the greatest probabilities of success. But the inquiry was not altogether confined to uric acid: experiments were also made on the solubility of cystine, oxalate of lime, and phosphatic calculi.

On the Solvent Treatment of Uric Acid by the Administration of Alkaline Salts by the Mouth.

The inquiry respecting uric acid, set out from two known data, viz.:

First.—That solutions of the alkaline carbonates exercise a solvent action on uric acid.

Second.—That the urine can be rendered alkaline from alkaline carbonates by the administration of certain salts by the mouth.

Starting from these data, a number of preliminary questions immediately presented themselves, which it was necessary to answer before proceeding to the more practical part of the inquiry. These were: 1. Whether is carbonate of potash or carbonate of soda[1] the better solvent for uric acid? 2. What is the best strength of solution to employ. 3. What is the effect of varying quantities of the solution on the results obtained?

Answers to these questions were sought by placing sections of uric acid calculi usually weighing about 100 grains, in 10 oz. phials, and causing currents of the different solutions, at blood heat, to pass over them at a regulated rate.

1. With regard to the *comparative solvent powers of carbonate of potash and carbonate of soda*, the experiments indicated clearly that potash dissolved uric acid more rapidly than soda. A solution of carb. potash, containing 30 grains to the pint, dissolved daily 11.9 per cent. of a uric acid calculus; while a solution of carb. soda of equal strength, dissolved only 10.3 per cent. The potash salt possessed a further advantage in its wider range of solvent power with the stronger solutions. This latter point will be better understood after the effects of solutions of different strength have been considered in the next paragraph.

2. The *Strength of the Solution* was found to affect its solvent capacity more than any other condition. It soon became apparent that only very weak solutions could yield any useful results. The greatest solvent power was found to reside in solutions containing from 40 to 60 grains of carbonate to the imperial pint. Below this strength, the power of the solutions gradually declined, until, with solutions containing less than three grains to the pint, the solvent power only slightly exceeded that of ordinary water. On the other hand, with solutions above the strength of 60 grains to the pint, dissolution was impeded, and

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[1] The experiments were principally directed to ascertain the effects of the alkaline *carbonates*, because all salts which have the power of alkalizing the urine to a useful degree, appear in the urine as carbonates. A number of other salts were however tried, viz., neutral and alkaline borates, phosphates, and soaps.

finally arrested, by the formation of a white crust or coat of alkaline biurate on the surface of the calculus. With a solution of 80 grains to the pint, this biurate crust was loose and easily detached, like a layer of whitewash; but with a solution of 120 grains to the pint, the crust was tenacious and adherent, and very little dissolution took place with carbonate of potash, and none at all with carbonate of soda. With solutions of 160 and 240 grains to the pint, there was no less of weight with potash or soda; the fragments became invested with a thin tough coating of white biurate, resembling white paint, which put a stop to all solvent action.[1]

The following table exhibits the results obtained with solutions of carbonate of potash of varying strength:

Strength of solution.	Daily average loss of weight.
240 grains per pint	0 per cent.
160 "	0 "
120 "	3.0 "
80 "	9.8 "
60 "	20.2 "
40 "	15.6 "
30 "	11.9 "
20 "	11.0 "
10 "	6.5 "
5 "	6.0 "
2½ "	2.8 "
1 "	1.2 "

The quantity of solution permitted to flow over the stone was, generally, six pints in the twenty-four hours.

3. It was at first supposed that the *quantity of the solution* permitted to flow over the stone would greatly influence the rate of dissolution; but on actual trial, the effect of quantity within the limits necessarily imposed by the capacity of the kidneys to eliminate fluids, proved to be comparatively unimportant. In order to obtain comparable results, different quantities of a solution of uniform strength were passed over the same stone on successive days.

A solution of carbonate of potash containing thirty grains to the pint, gave the following results:

Daily flow.	Daily loss of weight.
15 pints	13.0 per cent.
8 "	15.0 "
6 "	10.2 "
4 "	9.5 "

A flow of even one or two pints per day, with a solution of suitable strength, produced a copious dissolution. Two pints of a solution of carbonate of potash, containing forty grains to the pint, caused a daily dissolution of 17.1 per cent.

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[1] For further information regarding this white coating of biurate, *see* an abstract of a paper by the author in the Trans. of the Brit. Assoc. for the Adv. of Science for 1861; also Beale's Archives for 1862.

4. With regard to the *absolute rate of dissolution* attainable, the experiments opened out an inviting prospect. The solutions of maximum solvent power dissolved from ten to twenty per cent. of the calculi in the course of twenty-four hours. If results approaching these could be obtained in the living body, a little consideration will show that such an impression could be made on a uric acid concretion, in a few weeks or months, as would either entirely dissolve it, or reduce its dimensions to a point which would enable it to escape spontaneously by the natural passages.

Having disposed of these preliminary inquiries, the next points to be ascertained were: the best way of alkalizing the urine, so as to impart to it an alkalescence corresponding to that of solutions of carbonate of potash of maximum solvent power: also to examine the actual effect of alkalized urine passed over uric acid calculi, in a phial, at blood heat.

5. The most convenient *way of alkalizing the urine* was found to consist, in giving frequently repeated doses of the acetate or the citrate of potash. Both these salts are extremely soluble; they are well borne by the stomach; they do not interfere with digestion nor occasion purging. Weight for weight, the two salts were found to possess nearly equal alkalizing powers. With some individuals the acetate agreed better that the citrate; with others the converse was the case.

In order to maintain the urine at a degree of alkalescence that should correspond to the maximum solvent power of solutions of carbonate of potash (*i. e.*, an alkalescence equal to about 50 grains of carbonate to the pint), it was found necessary, in adults, to administer from 40 to 50 grains of the acetate or citrate of potash, dissolved in three or four ounces of water, every three hours.

It was found quite impossible to maintain the urine at an *absolutely constant* degree of alkalescence, however short the intervals at which the dose was repeated. The activity of the kidneys oscillates from hour to hour; at one time the urine is secreted abundantly and dilute, and then the degree of alkalescence necessarily falls; at another time it is secreted more scantily and more concentrated, and then the degree of alkalescence rises. When, however, the above dose was exhibited with regularity, every second or third hour, the oscillations rarely passed an alkalescence equivalent to 20 grains to the pint, on the one hand, and 80 grains to the pint on the other; and, as a rule, the alkalescence ranged between 35 and 60 grains to the pint—which corresponds, sufficiently exactly, with the maximum solvent power of a solution of carbonate of potash in water.

6. When urine, alkalized by the internal administration of these salts, was passed over the surface of uric acid calculi, at blood heat, the calculi were found to undergo solution at the mean rate of 12½ grains in the twenty-four hours.

In performing this experiment it was found that, unless the calculus and the phial were frequently cleansed by immersion in water, the urine became ammoniacal, and the calculus became covered over with a crust of the mixed phosphates, which speedily put a stop to the solvent action of the alkalized urine. An important practical deduction flowed from this, viz., *that when an ammoniacal state is developed, the solvent power of alkalized urine is entirely nullified by the deposition of the mixed phosphates on the surface of the calculus.*

The urine of patients taking full doses of the citrate or acetate of potash, is generally clear, and shows no tendency to deposit, even on standing. But this is not invariably the case; it is sometimes turbid from deposition of the amorphous phosphate of lime. Two conditions seemed specially to favor this deposition, namely, the febrile state, and the digestion of a heavy meal. The amorphous phosphate is not unfrequently deposited, as we have already seen (p. 78), after a meal, in healthy persons who are not taking any alkalizing medicines; the circumstance is, therefore, not to be regarded as an unnatural or hazardous one. It is, further, to be borne in mind, that the amorphous phosphate differs essentially from the mixed phosphates thrown down in an ammoniacal urine. The former is a loose flocculent substance, which shows no tendency to aggregate into concretions; the latter, on the other hand, is partly crystalline, and speedily encrusts any object brought into contact with it. The establishment of this distinction disposes of one objection which has been urged against alkaline solvents.

It now remains to bring forward illustrations of the application of the solvent treatment in practice; to distinguish the cases in which the treatment is applicable; to lay down directions for carrying it out effectually; and, finally, to examine some of the objections which have been urged against its employment.

7. *Illustrations* of the practical employment of alkaline solvents may be divided into cases of *renal* calculi, and cases of *vesical* calculi.

One of the first rational attempts to treat *renal* gravel of uric acid by alkaline solvents, was made by the celebrated Mascagni in his own person. He gives the following account of his case in the "Memoirs of the Italian Society for 1804:"

> I had been subject for several years to pains in the lumbar regions, and I voided from time to time gravelly concretions of a yellow-ochre

and brick-red color. Knowing that gaseous alkaline fluids had been used in such cases, I took some on several occasions with benefit. I imagined I could obtain greater effects with carbonate of potash.

In the months of August and September, 1799, having been obliged to lead a sedentary life, I was cruelly attacked with pains in the kidneys, and I voided a considerable number of small concretions, some of which were large enough to be regarded as veritable calculi. They were reddish and crystalline; they were deposited at the bottom of the vessel each time I made water, and I could see their glistening facets through the transparent urine. I was also subject to an excess of acid in the stomach, which was perceived in the mouth. I examined my urine and found in it a free acid, which, as well as the concretions, I recognized as consisting of uric acid.

Having thus assured myself of the nature of the concretions I was voiding, I resolved to make use of the carbonate of potash and to observe the result. I took the first day about a drachm, one-half in the morning fasting, and the other half in the evening. I dined at one o'clock in the afternoon. This salt dissolved in ten ounces of water had very little taste, it caused no disturbance of the stomach or bowels; but as soon as I swallowed it, it occasioned a considerable disengagement of carbonic acid gas, which was felt in the mouth and discharged by the anus.

The second day I took two drachms, and the third day three drachms; and I continued this dose, dissolved in twenty ounces of water, for ten days. Before using the carbonate my urine was very acid, and intensely reddened blue litmus paper. On the second day the paper changed color very little, and none at all on the third day. The acid of my urine was therefore saturated. At this epoch the renal pains diminished, and I voided no more gravel with the urine. Afterwards the pains ceased entirely, the urine became less loaded, and *I recognized the potash in excess.*

I ceased to use the carbonate of potash, and for some months I voided no concretions. Being subsequently attacked with the same symptoms, I had recourse to the same remedy, and I obtained the same good effects. I have repeated this medico-chemical experiment every time I have felt the same inconvenience, and always with success. Two years have now elapsed since I voided any concretions, though I no longer make use of the potash.[1]

The following example from my own practice is not dissimilar:

In July, 1860, a stout middle-aged gentleman brought to me eleven small concretions, varying from the size of a pea to that of a large pin's head. He had voided these with the urine a few days previously; they were composed of uric acid. He stated that three years before he was attacked with renal colic, which subsided on the third day with the discharge of a small calculus by the urethra. From this period to the time of my seeing him, attacks of renal colic, terminating in the discharge of small brownish concretions, recurred with great regularity at intervals of three or four months.

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[1] Magendie, De la Gravelle, p. 85.

The urine was found to be acid and high-colored; the general health was somewhat impaired by his periodical sufferings.

In projecting the plan of treatment, it was considered that the patient had in all probability a number of similar concretions still lodged in his kidneys. The dissolution of these was the first object; the next was to prevent their formation in the future. Seeing the small size of the concretions it was thought that by keeping up a persistently alkaline state of the urine for a week or two, complete dissolution of them would be effected. With this view citrate of potash, in two-scruple doses, dissolved in half a pint of water, was administered every three hours for a fortnight. Afterwards the patient took a drachm of the same salt in a tumbler of water night and morning for a period of three months. As no recurrence of the renal pains took place, nor the discharge of any concretions, the medicine was discontinued; but the patient was instructed to take every night before going to bed a tumbler and a half of water. This practice he has continued up to the present time (October, 1864). There has been no return of the symptoms.

A considerable number of examples of the successful treatment of *vesical* calculi by alkaline solvents lie buried in the forgotten publications which appeared in this country about the middle of the last century, when the remedy of Miss Stephens made so great a noise. Some fifteen or twenty cases were also collected by Chevallier and Petit at a later epoch, when the question was resuscitated in France about the year 1840.

Most of these reports are vitiated by the absence of information as to the nature of the stone and the condition of the urine. At the former epoch (1740), urinary calculi were all supposed to be of the same nature, and that an unknown one. At the latter epoch the chemical composition of urinary calculi was, indeed, known, but some of the most important points in their development were misunderstood; urinary chemistry was still in its infancy; and the same absurd pretension of universal efficacy was put forth on behalf of alkaline substances which swamped their reputation in 1740.

One of the best illustrations from the earlier records is supplied by Dr. James Jurin, who was himself the sufferer.

He was for many years subject to red gravel. At Christmas, 1740, he voided a small stone, after suffering four days from nephritic colic. In January and February following, he perceived unmistakable symptoms of stone in the bladder. These he describes at great length, and with remarkable clearness.

In March, he began to take lixivium of soap or soap-ley (a strong solution of caustic potash), in gradually increasing doses, until he reached the amount of an ounce or an ounce and a quarter daily. He took for a single dose one or two teaspoonfuls of the lixivium diluted with three-quarters of a pint of water. The soap-ley which he employed was "one-fifth part heavier than river water" (*i. e.*, its specific gravity was 1200, which is about three times as strong as the liq. potassæ of the London Pharmacopœia).

He continued this treatment for five months. On the 10th of July he voided a small smooth stone of the size of an oat, and of a reddish color. On the 27th of the same month he voided a second stone. On August the 6th he voided a third stone; and about the beginning of September a fourth.

All his symptoms now disappeared, and he discontinued the medicine; but in December he had a return of the vesical symptoms; he also noticed that his urine again furred the chamber-pot, and that he voided a little red gravel, as he had formerly done. He went back to the soap-ley, and in the course of a week parted with a small rough reddish stone. From that time he continued perfectly easy. He still took a couple of teaspoonfuls of the lixivium each day, and this he found sufficient to keep the urine from furring the utensil.[1]

The calculi in this case were undoubtedly uric acid, as may be learnt not only from their red color, but also from an experiment which Dr. Jurin made; he found that they dissolved in the alkaline ley and in lime-water.

Of the cases collected in France, I will only cite one. In Chevallier's essay, ten cases of the successful use of the bicarbonate of soda are recorded. Dr. Petit has contributed some half a dozen additional cases illustrating the effects of Vichy waters (which contain 44 grains of bicarbonate of soda to the pint).

M. de L——, fifty-one years of age, was sounded by Leroy d'Etiolles, who found a stone in the bladder. This he believed to be not large, and suitable for crushing. The patient, however, went to Vichy, and drank the first day seven or eight glasses of the waters. The next day he took fifteen glasses, and the urine, which was previously very acid, became constantly and strongly alkaline. In a few days he took twenty-two and twenty-four glasses. The symptoms, which were before severe, now subsided more and more, and after seventeen days of treatment he voided a smooth uric-acid concretion which bore evident traces of dissolution. From this moment he continued wholly free from symptoms, and was able to take violent equestrian exercise without the least inconvenience.[2]

The causes which led to the discredit and final abandonment of the alkaline treatment, in spite of the large mass of evidence in its favor, are now easy to understand. The most important of these was the erroneous claim to universal applicability set up for it by its advocates. My experiments prove unequivocally that it is wholly powerless in all cases where the urine is ammoniacal; also in all cases of oxalate of lime calculi, and in every variety of phosphatic calculi. No benefit can be derived from it except in cases of uric acid calculi, and in these, only where the urine has *not* become ammoniacal. The indiscriminate use of the treatment, therefore, could only result in disappointment. Further, the treatment was carried out in a very imperfect manner. In the earlier period (1740), alkaline substances were given in the form of soap, calcined egg-shells, lime-water, or solutions of caustic potash—all of them nauseous to the taste, apt to derange the stomach, and difficult to administer in sufficient doses to prove efficacious. In the later period (1840), Vichy waters were chiefly relied on. These contain soda, which, as we have seen, is an inferior solvent to potash; and the great dilution of the remedy in the Vichy waters must seriously impair its power.

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[1] The record of this case is bound up with Rutty's Observations on Joanna Stephens's Medicine for the Stone. Lond. 1742. Another good case is related by Whytt, in his Essay on Lime-water, Edin., 1752; and a third, in which the successful result is vouched for by a post-mortem examination made seventeen years afterwards, is recorded in the Philosophical Transactions for 1745, by Dr. Pringle.

[2] Dr. Ch. Petit, Du Mode d'Action des Eaux Minérales de Vichy, p. 272.

My own experience of the .alkaline treatment in vesical calculi was gathered before some important points were understood, which later inquiries have made clear to me.

My *first* case was one of uric acid calculus, and in every way suitable for the solvent treatment; but it was carried out very imperfectly, and was not persevered in sufficiently long to effect complete dissolution. The patient was a boy, four years of age, admitted into the Manchester Infirmary, December 1, 1858. The urine was acid, but did not deposit any crystalline sediment. He was placed under the influence of the tartrate of potash and soda (Rochelle salt), in the doses, at first, of twenty grains, and afterwards of thirty grains, dissolved in from four to six ounces of water, every two hours. The treatment was continued for six weeks. The urine was thereby rendered very freely alkaline. At the end of this period the sound still disclosed the presence of a stone, and the operation of lithotomy was accordingly performed by Mr. Southam, with perfect success. Two calculi were extracted, which together weighed only twenty-two grains; they were composed of pure uric acid, and their surfaces were perfectly smooth, and polished like river pebbles, without a particle of phosphatic incrustation.

My present experience enables me to point out two errors in the plan of treatment followed in this case. In the first place, the quantity of fluid in which the salt was dissolved was much too large; and in the second place, the salt used had too feeble an alkalizing power. Rochelle salt, on account of its large proportion of water of crystallization, has less alkalizing power by more than one-third than an equal weight of the citrate or acetate of potash.

Notwithstanding these drawbacks, it is not possible but that a considerable amount of dissolution had taken place. The urine was kept constantly, though feebly, alkaline for six weeks; there was no carbonate of ammonia developed in it, and no trace of phosphatic deposit on the stones. These are conditions in which, as my experiments prove, uric acid *must* undergo solution. The two calculi when extracted weighed only 22 grains; and yet one or both of them must have existed in the bladder for a period of three years, for the symptoms of vesical calculus had been distinctly noticed for so long. It is scarcely conceivable that these stones had not attained, in this length of time, a greater magnitude than they possessed when extracted; and it seems not too much to suppose that had the treatment, imperfect though it was, been persevered in for another week or fortnight, the size of the concretions would have been sufficiently reduced to permit their escape spontaneously by the urethra.

My *second* case was a boy, aged twelve, an inmate of the Manchester Children's Hospital, under the charge of Dr. Borchardt and Mr. Smart, who kindly permitted me to direct the treatment. The urine was acid; it contained a little pus, and had an inordinate tendency to deposit uric acid crystals.

On September 19, 1860, the patient was directed to take twenty grains of the acetate of potash in two ounces of water every three hours. This treatment was continued for thirty-four days; the urine was rendered thereby continuously alkaline. At the end of thirty-four days, the stone, being still found on sounding, was successfully extracted by Mr. Smart.

The calculus weighed 180 grains, and its form was a flattened oval; it was found to be composed of alternating layers of uric acid and oxalate of lime; and its surface presented a most peculiar appearance, which furnished an interesting and irrefragable proof of the solvent action of the alkalized urine on the uric acid layers of the stone.

The outermost layer consisted of uric acid, and over the larger circumference of the stone it had a thickness in its deepest parts of about a line and a half; but on the flattened surfaces the uric acid was dissolved away, and the subjacent layer of oxalate of lime cropped through it to a considerable extent. On one side the exposed patch of oxalate was as large as a sixpence, and presented the ordinary tuberculated appearance and dark brown color of a mulberry calculus. On the opposite side two islets of oxalate were uncovered, each about the size of a large split-pea. Surrounding the exposed patches of oxalate were found the remnants of a thinner, more superficial, and incomplete layer of oxalate of lime. The irregular patches of this latter layer occupied a higher level than the surrounding surface of uric acid; and here and there little elevations of uric acid could be seen surmounted with a shield of oxalate of lime. These elevations were partially undermined; the uric acid had been attacked by the solvent, and the protecting shield of oxalate of lime was in process of being thrown off by the gradual melting of its support.

The general surface of uric acid had a characteristic water-worn appearance. There were no minute mamillations such as usually stud the surface of uric acid concretions; but the surface was undulating, and the hollows and intervening ridges were perfectly smooth. No trace of phosphatic deposit existed on any portion of the stone.

Complete solution of the calculus was not possible in this case. A concretion composed of a uniform mixture of uric acid and oxalate of lime, was found to be attacked with considerable facility, by a solution of carbonate of potash, in the phial; and the present specimen shows that thin and incomplete layers of oxalate of lime may be undermined and disintegrated by alkalized urine; but if the stratum of oxalate be complete, and entirely invest the stone, it puts an absolute bar to further solvent action. This was the case in the instance before us. The partially uncovered layer of oxalate of lime surrounded the entire stone; and as soon as the dissolution of the superincumbent layer of uric acid had been completed, no further diminution of size could have taken place.

The treatment was not carried out in this case as efficiently as it might have been. The dose of the acetate should have been nearly double; this would have considerably more than doubled its solvent effect. The alkalescence of urine produced in a boy of twelve by twenty grains of the acetate every three hours is but feeble, and does not approach the highest solvent power capable of being imparted to the urine.

My *third* case was a boy of six, admitted under my care into the Manchester Infirmary, on January 27, 1862. The urine was acid, and singularly free from pus, blood, and other organic elements. He was placed under the influence of citrate of potash—at first, in the dose of twenty grains in six ounces of water every two hours. This was speedily raised to twenty-five grains, and continued with great regularity for two months. At the end of this period the dose was raised to thirty grains, given two-hourly in six ounces of water, and continued for a month longer. At the end of the third month the stone was still felt on sounding. The patient was then transferred to the care of my colleague, Mr. Southam, who successfully extracted the stone by the lateral operation. It proved to be a fine specimen of mulberry calculus, excessively rough on the surface, and not bearing the slightest traces of dissolution. Not a particle of phosphate existed on its surface. When sawn across a nucleus of uric acid was displayed. The outer crust of oxalate of lime was about a line and a quarter thick.

The solvent treatment was carried out with undoubted efficiency in this case, for the space of three months; but, of course, wholly in vain, owing to the impenetrable layer of oxalate of lime with which the stone was invested. The only defects which my later experiments enable me to point out, were the unnecessarily large amount of liquid administered and the unnecessarily frequent repetition of the dose. If the same dose had been given in half the quantity of water, and repeated every third hour, an equal effect on the urine would have been produced.

These three observations permit a deduction of great importance to be drawn from them, namely, *that a continuously alkaline state of the urine does not determine any precipitation of the earthy phosphate on the stone, so long as the urine is free from ammoniacal decomposition.*[1]

8. *Discrimination on the Cases in which the Solvent Treatment is, and is not, Applicable.* The first and most general limitation is:

A. *The Solvent Treatment is Inapplicable to all Cases in which the Urine is Alkaline.* The loss of the acid reaction of the urine in calculous cases, is due, in the overwhelming majority of cases, to ammoniacal decomposition from vesical catarrh. This state of the urine determines the precipitation of a phosphatic crust on the surface of the stone, and withdraws it completely from the influence of alkaline solvents.

B. *When the urine is acid*, the case may be regarded as *primâ facie* suitable to the solvent treatment; but there are still numerous limitations which reduce the cases really suitable within a much narrower compass.

(*a*) In the first place, *all those cases are excluded in which it is known or strongly suspected that the stone is composed of oxalate of lime.* This is sometimes ascertained from the patient having previously voided concretions of oxalate of lime; sometimes the character of the urine yields indications of the nature of the stone; if it deposit on cooling an abundant sediment of octahedra, or dumb-bells, the strong inference is that the stone is composed wholly or in part of oxalate of lime.

(*b*) When the examination of the urine and the previous history of the patient give *no indication of the nature of the stone*, we are left in doubt (supposing the urine to be acid) whether the calculus is composed of oxalate of lime or uric acid, or of alternating layers of these two substances. There are no data at hand to form an opinion as to the probabilities here involved. Different countries, and even different districts of the same country, show considerable diversities in the relative proportion of uric acid and mulberry calculi. Renal calculi also differ essentially in regard to this point from vesical calculi. The former are generally composed of a single substance; and in about five-sixths of the cases this is uric acid. The latter, if they have sojourned any considerable time in the bladder, are frequently composed of two or more substances arranged in alternate layers.

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[1] Ten years ago I was consulted by a gentleman, aged 67, who, on sounding, by myself and a colleague, was found to be suffering from stone in the bladder. The urine was loaded with uric acid crystals. He was put on a full course of the bicarbonate of potash for a period of nearly six months. The symptoms slowly subsided; no stone was voided, but the patient has since remained, and is now (January, 1876) perfectly free from symptoms of stone or of any irritation about the bladder.

In cases of *renal calculi* the patient should evidently have the benefit of the doubt. No other treatment than that by alkaline solvents is open to the choice of the practitioner; and if the calculi should be composed of oxalate of lime, the alkaline treatment will not aggravate, if it do not ameliorate, the state of the patient.

In cases of *vesical calculi* the question stands differently. The solvent treatment comes here into competition with the mechanical methods of lithotomy and lithotrity, which long experience have stamped with success. It is no longer a question of the mere possibility of removing a calculus by means of solvents, but of doing it with less risk than by lithotomy or lithotrity.

Future experience can alone decide, whether it is better in cases of this class (where the nature of the stone is quite uncertain), to consign them at once to the operating table, or to give a preliminary trial to the solvent treatment. It would appear from the cases reported in the preceding pages, that patients who have undergone such a trial may be afterwards transferred to the surgeon with undiminished chances of a successful operation. Probably the most advantageous course to follow, if the stone be a small one, would be, to try the solvent treatment for a limited period—for six weeks or two months—and, if unsuccessful at the end of that time, to proceed without further delay to operation.

(*c*) *When the stone is known to be a large one*, the solvent treatment should not be attempted. The presence of a large stone in the bladder is itself a perpetual source of danger; and the larger the stone, the greater the probability that it contains one or more layers of oxalate of lime, which will resist the solvent. The length of time which a stone above the weight of an ounce would require for dissolution, also detracts greatly from the advantages of the solvent treatment, as compared with the swifter, though less safe method of lithotomy.

(*d*) The cases of vesical calculi *which are especially suitable for the solvent treatment, are those in which it is known or strongly suspected that the concretion consists of uric acid, and has not yet attained any great size.* It not unfrequently comes to pass that an individual who has previously, at divers times, spontaneously voided small uric acid calculi, becomes afterwards the subject of vesical calculus. If such a case come under treatment soon after the first appearance of symptoms of stone in the bladder, it is one peculiarly promising for the solvent treatment. The stone is sure to be small, and it is almost certain to be wholly composed of uric acid. A dissolution of twenty or thirty grains would reduce the stone sufficiently to enable it to traverse the urethra. A more rational, safe, and certain plan of treatment is scarcely conceivable in any disease.

(*e*) It is probable that the solvent treatment judiciously carried out, will prove a useful *adjunct to lithotrity*. It is, however, essential to its employment that no vesical catarrh, with ammoniacal decomposition of the urine, ensue after the operation. If the urine maintain its acidity after the stone is crushed, and if the fragments discharged prove to be uric acid, then the solvent treatment might be expected to act advantageously by obviating the inconvenience and danger of repeated sittings.

To sum up in the affirmative: the solvent treatment is only applicable in those cases of vesical calculi in which *the urine is acid; the stone not large; its composition known to be uric acid or strongly suspected to be such.*

9. *Rules for Carrying out the Solvent Treatment.* The action of alkalized urine is essentially slow; quick solution, by any manner of applying it, is impossible. To make up for this defect, its operation must be continuous and incessant. To rest content with alkalizing the urine for a few hours each day, is not only to reduce the solvent effect to an insignificant quantity, but, sometimes at least, to nullify it altogether. I have known urine kept continuously alkaline by acetate of potash for many successive days, recover its acidity and deposit uric acid within a few hours of the latest dose. It is also of great importance not only to keep the urine continuously alkaline, *but to keep it alkaline to a certain degree.* The experiments described at p. 318 prove that solutions with an alkalescence below three grains of carbonate of potash to the pint, have scarcely a greater effect on uric acid calculi than simple water. A feebly alkalized urine acts so slowly, that (in cases of vesical calculi) the delay incurred counterbalances the safety of the treatment as compared with mechanical means, and robs it of the preference which it might otherwise deserve.

To secure a continuous alkalescence, the dose should be repeated at intervals of not less than three hours, and it should be given with rigorous regularity during the waking hours. A dose should be taken the last thing before retiring to rest, and another in the course of the night. Of course, a patient should not be disturbed from sleep in order to take a dose of medicine; but patients with vesical calculi, scarcely ever are able to pass the night, without awaking spontaneously once or more to empty the bladder.

The best salts for administration are the acetate and citrate of potash. Of the former, the dose for an adult should be from 40 to 60 grains dissolved in 3 or 4 ounces of water; for children, the dose should range from 20 to 30 grains. The citrate (anhydrous) has nearly the same alkalizing power as the acetate. The citrate of potash of the shops is of uncertain strength, and often exceedingly impure. The best plan is to prepare the solution directly from the crystallized bicarbonate of potash and crystallized citric acid. The bicarbonate, when saturated with citric acid, forms almost exactly its own weight of anhydrous citrate; so that when 40 grains of bicarbonate of potash are saturated with the proper quantity of citric acid, there result 40 grains of citrate of potash.

The following prescription yields a solution containing one drachm of the citrate in each fluid-ounce:

℞.—Potass. bicarb.	ʒxij.
Acid. citric.	ʒviij. gr. xxiv.
Aquæ	ad ℥xij.

The dose of such a solution for an adult, is 6 or 8 fluid-drachms mixed with 3 or 4 ounces of water; and for children, 3 to 6 fluid-drachms diluted in the same proportion.

In conducting the treatment, it is essential that the freshly voided urine should be frequently examined. If at any time it shows signs of ammoniacal decomposition the treatment should be suspended. The advent of this state is indicated by the offensive ammoniacal smell of the urine and the increase of pus and flaky matter in it. As long as the urine continues sweet *when voided*, no fear need be entertained of the deposition of the mixed phosphates on the surface of the stone.

10. *The objections* urged against the alkaline treatment have been chiefly three:

(*a*) It has been alleged, that by rendering the urine alkaline, there is danger of the precipitation of the phosphates on the surface of the stone. The facts advanced in the preceding pages dispose of this objection completely. If there be ammoniacal decomposition of the urine, the phosphates are deposited whether alkaline medicines be given or not, and the concretion goes on increasing; but if the urine be alkaline solely from fixed alkali, not a particle of phosphatic deposit takes place.[1]

(*b*) It has been said that the natural reaction of the urine is acid; and therefore, that to render it alkaline is to introduce an unnatural state, which cannot fail to act deleteriously on the general health. In a state of fasting the natural urine is doubtless always acid; but the researches of Dr. Bence Jones, fully confirmed by my own (*see* p. 77), show that the urine is normally alkaline (from fixed alkali) for several hours daily, after meals, in many, if not all healthy persons. So that the maintenance of an alkaline reaction of the urine by fixed alkali is by no means so unnatural a state as some have supposed.

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[1] A want of knowledge of the essential difference between urine alkaline from fixed alkali and urine alkaline from carbonate of ammonia, runs, like a thread of error, through the elaborate argument of Civiale, in his chapter on the "Dissolution of the Stone." *See* chap. iv. of his work, Du Traitement Médical de la Pierre.

(*c*) Alkaline substances, it is urged, impair digestion. This objection was valid against the ruder methods of alkalizing the urine formerly employed. But the acetates and citrates have no such effect. The introduction of these salts (and the bicarbonate) in recent times for the treatment of articular rheumatism, has afforded an immense field for watching their effects. Indeed, the solvent treatment here recommended is identical with the alkaline method of treating rheumatism, except that the dose is administered in a somewhat more dilute form. In the last twenty years I have employed the bicarbonate, the acetate, and the citrate of potash, both in private and public practice, in doses of four, six, and eight drachms in the twenty-four hours, in a very large number of cases. The majority were cases of articular rheumatism; the remainder embraced a variety of slighter and more severe disorders—skin diseases, emphysema, diabetes, acute Bright's disease, etc. The urine was kept continuously alkaline for periods varying from a fortnight to three months, and in no instance were deleterious effects observed. In one patient with pulmonary emphysema, the urine was kept uninterruptedly alkaline for fourteen weeks, with marked improvement of the general health and steady increase of weight. In short, the acetate and citrate of potash have appeared to me about as harmless as so much sugar.

The evidence of the Rev. W. Vernon Harcourt on this point is worth quoting. He was for several years the subject of stone in the bladder; but, owing to his great age, and the slight inconvenience he suffered, operative procedures were not considered desirable. On the suggestion of Mr. Spencer Wells and myself, he resolved to try the alkaline treatment, more with the purpose of keeping the growth of the stone in check, than with the hope of removing it altogether. Mr. Harcourt has published an interesting account of his case, which he made the subject of a chemical study. I have already quoted his improved method of estimating the amount of uric acid in the urine (*see* p. 90). Speaking of the effect of the alkaline treatment on himself, he says:

"My experience of the effects of citrate of potash, not exceeding 300 grains taken in 24 hours, and producing an alkalinity equalizing from 20 to 25 grains of carbonate of potash (per pint), continued during three months, has convinced me that no sensible disadvantage to health need be feared from such a course; and this is the experience of a man eighty years of age, who has been for some years an invalid. Neither during nor since the treatment has any irritation of the bladder been felt, and the urine has been for many months perfectly clear; it was never ammoniacal or albuminous."[1]

On the Solvent Treatment of Uric Acid Calculi by Injections into the Bladder.

It has been conceived that considerable advantage would be gained, in cases of vesical calculi, by injecting the solvent directly into the bladder, in a continuous stream, by means of a double-current catheter. The advantages chiefly counted on were: the use of stronger solutions, and the employment of a greater mass of the solvent. In the case of uric acid calculi, numerous experiments undertaken by myself show clearly that these advantages are illusory.

The mode of proceeding which I adopted, was to place a section of a uric acid stone in a ten-ounce phial, and to pass over it at blood heat, a current of the solvent as large as the capacity of the urethra might be supposed to permit. The current was kept up for two or three hours continuously.

From experiments already recorded at p. 318, the maximum solvent power of the carbonated alkalies is ascertained to lie in solutions containing about 50 grains to the pint. A solution of *carbonate of potash* of this strength was passed over a fragment of uric acid weighing 57 grains, at the rate of forty-two pints per hour, for a period of three hours. The result was a dissolution at the rate of two grains per hour. This result, insignificant as it is, could probably not be approached in the living bladder on account of the mechanical difficulties to be overcome.

Solutions of the following substances were also tried in a similar manner, namely, *borax*, *borax with liquor sodæ*, *double borate of potash and soda*, *common phosphate of soda*, *basic phosphate of soda*, and *potash soap;* but their solvent effects did not reach beyond a loss of weight of one or two grains in the hour.

Lime-water in a continuous current, at the rate of 30 pints per hour, dissolved a fragment weighing 86 grains, at the speed of one and a half grain per hour.

Seeing the very small results thus obtained, I proceeded to try the *caustic alkalies*, which are the most powerful known solvents of uric acid. But solutions, such as could be borne by the living bladder, of liquor potassæ and liquor sodæ (60 and

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[1] Med. Times and Gaz., 1869, ii. 484.

120 minims to the pint), did not dissolve more than about two grains per hour.

The general conclusion from these experiments[1] is, that under the most favorable conditions, and with the most effective solvents capable of being borne by the living bladder, no greater dissolution than one or two grains per hour can be accomplished in the case of uric acid calculi. In actual practice the conditions would necessarily be much less favorable than in an experiment performed in the laboratory. A little consideration is sufficient to show that these results hold out no prospect of any useful practical application.

SOLVENT TREATMENT OF CYSTINE CALCULI.—Cystine is soluble both in the carbonates of the fixed alkalies and in the mineral acids. It may therefore be attacked, when existing as a calculus in the bladder, either by alkalizing the urine, as in the solvent treatment of uric acid, or by injecting acid solutions into the bladder.

Two experiments were performed with a view of testing the solubility of a cystine calculus in a solution of carbonate of potash containing 40 grains to the pint. The mean result, with a daily flow of three and six pints, showed a rate of dissolution equal to 20 per cent. of the weight of the stone in twenty-four hours.

I have recently (1883) had an opportunity of testing clinically the solvent power of alkaline remedies in a case of cystine calculus. The results were wholly disappointing. No impression whatever was made on the concretion—although the treatment was carried out resolutely for several months. And I was surprised to find that the patient's urine, even when rendered fully alkaline by the administration of citrate or bicarbonate of potash, still contained crystals of cystine floating in it. This indicated not only that the treatment was not exercising any solvent action on the concretion, but that the conditions for its growth and enlargement still persisted in spite of the alkaline reaction of the urine. I attributed this failure to the concurrent presence in the urine of carbonate of ammonia. For the carbonate of ammonia, unlike the carbonates of potash and soda, does not dissolve cystine—on the contrary it precipitates it from its alkaline solutions. The stone was a very large one, and it subsequently was removed by Sir Henry Thompson by the suprapubic operation—and the patient made an excellent recovery.

SOLVENT TREATMENT OF OXALATE OF LIME CALCULI.—In the case reported at p. 326, alkalized urine flowed over the surface of a mulberry calculus for three months without producing the slightest show of solution. I also found that a solution of carbonate of potash, containing 40 grains to the pint, passed over a mulberry calculus at the rate of six and eight pints in the twenty-four hours, had not the slightest solvent effect.

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[1] The experiments here referred to are more fully described in the author's paper in the Medico-Chirurgical Transactions for 1865.

Better results, it was conceived, might be obtained by a solution of dilute nitric acid (which is the best solvent of oxalate of lime), employed so as to imitate injections into the bladder. A solution, containing 120 minims of the concentrated acid to the pint, was passed over a mulberry calculus weighing 53 grains, at the rate of 24 pints per hour; and yet only half a grain was dissolved in an hour. We may conclude, from these experiments, that oxalate of lime calculi are unassailable by solvents applied in any known method.

Solvent Treatment of Phosphatic Calculi.—Phosphatic calculi were found quite unimpressible, as might have been expected, to solutions of carbonate of potash. Far more promising results were obtained by dilute nitric acid, used so as to imitate injections into the bladder. A solution containing 60 minims of the commercial acid to the pint, was passed at blood-heat over a phosphatic stone weighing 153 grains, at the rate of 36 pints per hour. The loss of weight which followed amounted to 21 grains per hour. A modification of this proceeding was successfully employed, as is well known, by Sir B. Brodie, in actual practice. My colleague, Mr. Southam, has also tried the same method, and with the best results. A stone had been repeatedly crushed with the lithotrite; but fresh phosphatic concretions formed in the bladder as fast as the old ones were broken up; and it was found impossible completely to clear the bladder. In this difficulty an injection, containing two drachms of dilute nitric acid to a pint of water, was practised every day or every second day. In the course of a short time the old fragments were completely dissolved, and the formation of new ones prevented. This method is evidently capable of wider application than is now made of it by surgeons.

CHAPTER IV.

CHYLOUS URINE.

THE disorder named by Prout chylous urine, is mainly a disease of tropical climates. It prevails endemically in the Mauritius, Isle of Bourbon, West Indies, the Brazils, and India. The majority of the cases met with among Europeans are found among sailors, merchants, colonists, and others who have passed a portion of their lives in one of the above-named countries.

In this disorder the urine is usually white and opaque, like milk; sometimes it has a faint rose tint, from a slight admixture of blood; and sometimes it is mixed with blood in clots.

On standing awhile, it sets spontaneously into a trembling coagulum, which after a time redissolves, and breaks up into flaky clots. Not unfrequently this coagulation takes place within the bladder, and occasions serious pain and difficulty in micturition. The milky appearance of chylous urine is due to the presence of a finely divided fatty or oily matter. This is thrown up as a creamy layer after the urine has stood some hours. When chylous urine is agitated with ether, the fat is dissolved, and the secretion assumes the transparency and color of healthy urine. The ethereal extract yields, on evaporation, a quantity of yellowish, solid or oily uncrystallizable fat, resembling that which is found in the blood. Chylous urine is invariably coagulated by heat and nitric acid. These united reactions indicate the presence of fibrine, fat, and albumen. Caseine, though specially looked for by many observers, has never been authentically found in chylous urine; nor is sugar usually found therein.[1] The ordinary ingredients of healthy urine are present in their usual proportion, unless there is some superadded disease. The specific gravity is generally below the average. When examined microscopically, chylous urine is found to contain a variable number of granular nucleated corpuscles, like those of mucus or chyle; and generally, but not always, red blood disks. The fatty matter almost invariably occurs in the form of excessively minute granules (resembling the molecular base of the chyle), which are not resolvable into visible globules

[1] Dr. Habershon observes glycosuria and polyuria in connection with chyluria. In this case the sugar persisted after the fatty matters had disappeared from the urine. Yet it was not merely a case of the fatty urine which is occasionally observed in diabetes, for the urine coagulated spontaneously (*see* Lancet, 1880, i. p. 171). Mr. Morison also has described a case in which sugar was present, and where the fat could be obtained in crystalline form. (Path. Trans., vol. xxix. p. 394.)

under the highest powers of the microscope. Occasionally, however, visible fat globules are found, as in the case recorded by Dr. Waters. Casts of the uriniferous tubes have never been found, though specially searched for by Bence Jones, Waters, Isaacs, Begbie, and myself.

Sometimes the urine is not chylous, but *lymphous:* that is, it contains albumen, and coagulates spontaneously, but the fat is absent, together with the opaque milky appearance which depends thereon. The coagulum in lymphous urine resembles calf's-foot or currant jelly.[1]

In this curious disorder the urine resembles in every particular a mixture of ordinary urine with variable quantities of chyle or lymph: and a strong probability exists, as will be presently seen, that chylous and lymphous urines are, in fact, such mixtures.

The unnatural ingredients—albumen, fat, and fibrine—vary considerably in their relative proportions. The following table presents an abstract of nine analyses of chylous urine by different authors:

	Quevenne. (Rayer, iii. 427.)	Rogers. Mean of 3 analyses. (Bird, p. 420.)	Bouchardat. (Annuaire, 1862, p. 201.)	Beale. (Archives, v. 1. p. 12.)	Bence Jones. (Phil. Trans., 1850.) Mean of analyses.	Dr. B. Edwards. (Med.-Chir. Tr., v. xlv. p. 217.)	A. Gamgee. (Ed. Med. J., Aug. 1862.)
Fatty matter . . .	1.90	1.10	1.30	1.39	0.79	0.99	0.20
Albumen	0.70	0.33	0.20	1.30	1.40	0.60	0.17
Normal solids of the urine.	2.30	4.71	3.73	2.57	2.88	1.68	3.04
Water	95.10	93.86	94.77	94.74	94.93	96.73	96.59
	100.00	100.00	100.00	100.00	100.00	100.00	100.00

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[1] Mr. Stocks, of Salford, sent to me, November 18, 1864, a man named Williams, aged twenty-seven, who had never resided out of England. In 1862 this man was the subject of lymphous urine for about a month. He was at that time suffering from an extensively distributed, and severe, cutaneous disease of an eczematous character. Mr. Stocks gives the following description of the urinary symptoms. 'There was great pain over the kidneys, in the perineum, and about the anus—defecation aggravating the latter much. No tenderness existed in the prostate. There was stillicidium urinæ, and frequent, painful, straining micturition—half an ounce of urine passing at once. Masses resembling pieces of tripe, about the thickness of a lead pencil, were pulled out of the urethra two or three times a day for about a week. The urine itself was clear, highly albuminous, and, when allowed to stand, coagulated spontaneously into yellow transparent masses, floating in the fluid part of the urine, exactly like half-melted calf's-foot jelly. These masses again became fluid in about twenty-four hours, leaving cobwebby fibres floating in the urine.'' When this man was seen by me he had lost the cutaneous eruption, and was able to follow his employment as warehouseman. Micturition was still unduly frequent; but the urine was free from fibrine and albumen. The only unnatural objects found after a careful microscopical examination were a few blood and pus corpuscles. Was this case an example of eczema invading the mucous membrane of the bladder?

The *course* of the disorder is marked by an irregularity and capriciousness which baffle explanation. The invasion is sometimes gradual; but more commonly it breaks out suddenly without previous warning or known cause. In other cases it comes on apparently after a fall or shock,[1] or in consequence of hard mental or bodily work. In a case seen by me the disease came on after parturition. Its further progress is essentially intermittent; but it rarely happens that the intermissions follow any regular rule. An attack may last a few days, a few months, or many years. The intervals between the attacks vary similarly; the disorder may go on intermittingly for two, three, or more years, then cease for ten or more years, and be again renewed. The suspensions and renewals are generally quite abrupt, sometimes more gradual. During the remissions the urine returns to a perfectly normal state. Sometimes the attacks observe a certain periodicity. In one case it is related that the urine always became chylous for eight days previous to menstruation; in another, the recurrence almost always preceded or accompanied attacks of epilepsy or erysipelas. In Mr. Pearse's case the urine became chylous when the patient was suckling her children, and ceased to be so shortly after weaning them. It has been observed in several instances that an intercurrent disorder, such a fit of the gout, hepatitis, carbuncles, inflammation of the lungs, severe ptyalism, has temporarily suspended the chylous condition of the urine. In other instances it dates its origin or renewal from some such attack.

There are also diurnal irregularities in regard to meals, exercise, and rest, which are inexplicably contradictory. As a rule, rest and fasting diminish or suspend the milkiness of the urine. In some cases the urine is chylous throughout the twenty-four hours: in others it is natural or lymphous on rising in the morning, and chylous during the remainder of the day, especially after dinner; in Mr. Cubitt's case (cited by Beale) the urine was never chylous during the day, but only on rising in the morning. In Ackermann's case the urine became perfectly natural when the patient lay on his right side, and immediately resumed it chylous character when he stood up. Dr. Bence Jones found, in a case observed by him, that meals and exercise had a marked influence on the state of the urine. Shortly after a meal the urine became chylous: if a patient fasted and took exercise, the urine was lymphous: if he fasted and remained perfectly tranquil, it became natural. Dr. Mackenzie observed in his case, that alterations in the meal-times produced alterations in the characters of the urine. Thus, the day urine almost completely coagulated and contained a considerable amount of blood, while the night urine did not form so large a coagulum, and contained less blood and was much more milky. An alteration of a few hours in the meal-times caused the characters of the day and night urines to approximate, while when the habits of day and night were completely reversed, the conditions of the urine were similarly reversed. In Mr. Dutt's case the urine voided during the day was clear and free from chyle, while that voided during the night and in the morning was deeply loaded with it.

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[1] In Egger's case the patient attributed her disorder to the mental and bodily shock sustained in a fearful railway collision, six months before the urine first became chylous.

ILLUSTRATIVE CASES.

The following abstracts of cases will convey an idea of the character of the urine and the capricious course of the disorders:

CASE 1.—A woman, æt. 25, was admitted into the Manchester Infirmary in the spring of 1868, suffering from chylous urine. She was born in this neighborhood, and had never lived out of the county. Seven months ago she was confined, and ever since the urine had been milky. Her general health on admission was good. The urine was more or less milky throughout the twenty-four hours—sometimes it resembled thin skimmed milk, and sometimes it had the appearance of rich new milk. It generally coagulated spontaneously on standing a few hours into a tremulous jelly, resembling ill-made blanc-mange. At other times no spontaneous coagulation took place, the milky fluid maintaining its diffluence unchanged for two days. It did not throw up, as a rule, any creamy layer on standing. Its specific gravity varied from 1012 to 1018. When fresh it was quite odorless. Under the microscope no visible oil globules were seen, but myriads of minute molecules. In addition to the molecules a number of cells were seen exactly resembling chyle corpuscles, and a few scattered red blood-disks. Crystals of triple phosphate appeared in a few hours. It coagulated freely both with boiling and with nitric acid, but acetic acid produced not the slightest change in it. When the urine was shaken up with an equal volume of anhydrous ether, the milky appearance was lost, but it did not become quite transparent; it changed to a yellowish-brown, with some degree of turbidity, and after a few hours a moderate deposit of chyle corpuscles subsided. The milky appearance was not removed by filtering through paper. No casts of tubes could be detected after a long search. Various remedies were tried by Dr. Morgan—under whose care the patient was—but without success. The patient left the Infirmary, and has been since lost sight of.

CASE 2.—A married woman, aged thirty, had been passing chylous urine about a year. Three specimens of her urine were submitted to Dr. Prout for examination, namely, one voided in the morning; another a little after the breakfast; and a third in the evening.

The first specimen, voided in the morning, consisted of a solid jelly-like mass or coagulum of a pale amber color. This coagulum was of an extremely delicate texture, and, on being submitted to a gentle pressure, or even allowed to drain, parted with a large proportion of a serous fluid of the color above mentioned, and at the same time became exceedingly reduced in bulk, and assumed the appearance of a red fleshy-looking mass of a fibrous texture, which on examination was found to have all the properties of the fibrine of the blood, mixed with a few red particles of the same fluid. The specific gravity of the serous portion was 1019. Its smell was very faintly urinous; reaction neutral; contained a large quantity of albumen.

The second specimen, voided after breakfast, resembled the first in its general character, but differed from it in some minor particulars. Thus, the serum was more of a whey color; the fibrous coagulum was less, but more compact and firm, and held entangled in its texture a large proportion of the red particles of the blood. The specific gravity of the serum was only 1012, and it contained a considerable proportion of albumen.

The third specimen, voided in the evening, after an early dinner taken about noon, so closely resembled chyle in all respects, that Dr. P. was doubtful, if it had been brought to him as a specimen of that fluid, whether he should have discovered the imposition. It consisted of a solid coagulum of a white color, and assuming the shape of the vessel, like blanc-mange. On being submitted to a gentle pressure and permitted to drain, the residual solid portion was, like that of the others, small in quantity, but whiter than the coagula of the other specimens. It was, however, intermixed with strings of a firmer consistence and of a red color. The serous portion was white and opaque like milk, and, on being heated and allowed to stand at rest for some time, threw up a substance on its surface resembling cream, and which, like cream, contained a considerable proportion of oily principle. Its specific gravity was 1017, and its smell was not urinous until it had been concentrated by evaporation; it was not coagulable by heat, though it contained abundance of albumen.

Dr. Prout had an opportunity of examining this woman's urine after fasting twenty-four hours. The coagulum was now much smaller in bulk, and seemed to contain more red particles. The serous portion was nearly transparent, and possessed in a considerable degree the color and other sensible properties of urine. It contained albumen and abundance of urea.

This woman died emaciated, after suffering from the disease twenty years. (Prout on "Stomach and Renal Diseases," 5th ed., p. 117.)

Case 3.—The patient was a sailor, a native of Bermuda, treated by Dr. Waters in the Liverpool Northern Hospital. The characters of the urine are thus described. When first passed, it is white, with rather a pink tinge. It resembles new milk in appearance and somewhat in smell. It is perfectly free from urinous odor. After it has been passed for a short time it coagulates into a tremulous mass exactly resembling blanc-mange. The coagulum sooner or later disappears, entirely or in part, leaving the urine altogether fluid or partly clotted. After the urine has been standing some hours, a distinct deposit of florid blood is found at the bottom of the vessel, and the mass of fluid above assumes a perfectly white color, showing that the pink appearance of the urine when first passed was due to the admixture of blood. There is, in addition to the blood-deposit, a deposit of a somewhat slimy character, having all the appearance of a mixture of pus and mucus. After standing some hours, a distinct thin layer of white fluid, exactly resembling cream, generally forms on the surface of the urine, the layer being thicker in some specimens than in others. The urine remains free from odor for some time, but at the end of three or four days it has a slightly urinous smell.

When first passed the urine is slightly acid or neutral, and soon becomes alkaline. Heat causes a precipitate of very fine particles. Nitric acid also produces a slight precipitate, but heat and nitric acid together cause a copious deposit. When boiled with liquor potassæ and sulphate of copper, there is no reduction of the copper to the state of suboxide. If the urine be agitated in a test-tube with an equal part of sulphuric ether and left to stand, a thin layer of fatty (?) matter is deposited on the surface of the urine and below the ether. The urine then becomes quite clear, and if removed by means of a siphon and boiled with nitric acid, a copious deposit takes place.

When examined under the microscope the urine is found to contain blood, pus, and mucous corpuscles, with a large number of small fat globules. Many of these last are very minute, while others are larger. No casts of the uriniferous tubes nor any other abnormal matters than those already mentioned, were found in the deposit. The thin layer of cream-like fluid before alluded to consists entirely of oil globules. (Dr. Waters, "Med.-Chir. Trans.," vol. xiv. p. 211.)

Contrasting with this description in the occasional absence of spontaneous coagulation and of visible fat globules, is the following account by Dr. Beale:

CASE 4.—The specimen of urine was passed in the morning. It was perfectly fluid, without any tendency to spontaneous coagulation, and had all the appearance of fresh milk. It had neither a urinous smell nor taste. Upon the addition of an equal volume of ether, it became pefectly clear.

Under the microscope the slight deposit which formed after standing some time, was found to consist of a small quantity of vesical epithelium, and some small slightly granular circular cells resembling chyle or lymph corpuscles. No oil globules could be detected on the surface of the urine or amongst the deposit, and the fatty matter, which was equally diffused throughout, was in a molecular or granular form. By examining the urine with the highest powers only very minute granules could be detected. These exhibited molecular movements. In this case the urine was not, however, always uncoagulable, occasionally it coagulated even within the bladder. This case is exceptional in the circumstance that the urine was never chylous, except on rising in the morning. During the rest of the day it was always perfectly natural. The patient was a native of Norfolk, and does not appear ever to have been out of England. (Beale, "Urine and Urinary Deposits," 3d ed., p. 300.)

CASE 5.—A clergyman, aged forty, born in Bermuda, consulted Dr. Bence Jones in 1852. Ten years before the urine became milky, and continued so for eight weeks; it then returned to its natural state without treatment. Five years after the complaint returned. The patient passed clots and semi-solid masses with some difficulty. The second attack lasted two or three months, and then the urine became perfectly natural, and continued so for the succeeding four years. At the end of this period the disease returned, and had continued ever since, with the exception of an interval of three weeks. When the patient came under the care of Dr. B. Jones, in 1852, the urine was milky, but it cleared with ether; it contained much albumen and some blood-corpuscles, but no casts of tubes; its reaction was acid, specific gravity 1025. The patient stated that bodily or mental exertion (such as preaching on Sunday) produced the most intense milkiness of the urine. Usually the urine was milky on going to bed; it was clear in the morning, until an hour after breakfast; the whiteness then increased according to the degree of exercise taken. He dined at one, and then, with rest, the urine became clear, and continued so until he took his afternoon walk, when the whiteness returned. He had tried all sorts of tonics, buchu, and iron, and had taken gallic acid on this and the previous occasions without advantage. (Bence Jones, "Med.-Chir. Trans.," vol. xxxvi. p. 91.)

CASE 6.—A lady, aged sixty-four, born in India, where she had resided for some years, came under the care of Dr. Elliotson. The urine became for the first time milky *nine years* after her return to England. It continued milky, in spite of various remedies, for about a year, when it suddenly resumed its natural appearance on the third morning after she had commenced to take a daily bath in the sea. The urine then remained clear for thirteen years (eighteen months of which were passed in India). At the end of this period she had a severe inflammation of the lungs, for which she was bled, and took calomel. In a month after this attack the urine again became chylous, and continued so for two years. At this time she suffered from a severe mental shock, and for the space of one month the milkiness of the urine was suspended; it then returned with as much intensity as ever. From this date the urine continued milky without one day's intermission for eleven years. The milkiness was then, once more, suspended for six weeks in consequence of a carbuncle; then it went on again for two years, when she had a second attack of inflammation of the lungs, which laid her up for six months. During this illness the chylous state of the urine was again suspended; but has since returned, and still continues (1857). The patient is a very stout person, and very nervous. All remedies have been unavailing. The disease has continued, with intermissions, for eight-and-twenty years. (Elliotson, "Med. Times and Gaz.," 1857, ii. 287.)

CASE 7.—An English gentleman, aged forty, a teacher of languages, settled in Rostock (Germany), came under the notice of Dr. Ackermann. In his youth, this gentleman had travelled for two years in the Brazils and Buenos Ayres. While in South America he suffered from a slight hydrocele,[1] which appeared on his return to Europe.

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[1] Query—Was not this supposed hydrocele an affection of the scrotal lymphatics, as in Carter's cases, to be presently described?

He had been settled in Rostock for eleven years, and was strong and very healthy. Midsummer, 1858, he took the measles, which left behind a slight bronchial catarrh. This catarrh lingered on till February, 1859; when one day he observed his urine to be milky. A few hours before this occurrence he felt out of sorts and shivered, but next morning he was well again. The urine, however, continued milky, and five weeks after, leeches were applied to the left loin on account of a tenderness which existed in that region. During their application the patient lay on his right side for two hours, and immediately on rising he made water, which, much to his own and his physician's astonishment, was perfectly normal, clear, and of a deep yellow color. Nevertheless, at the next micturition, the urine was found milky again. A few days after the patient repeated the experiment; he emptied the bladder, and lay for an hour on his right side, and again the urine appeared clear, and contained only traces of albumen. Similar results were obtained many times after the same experiment. If perfect rest on the right side was broken even for a few minutes, the urine was distinctly chylous at the end of the experiment. A counter-experiment, in which the patient lay on his left (instead of right) side, showed still more distinctly the effect of the posture on the right side. On the 6th of May the patient lay for an hour on his left side; but the urine which he made on rising was strongly chylous and contained blood. At a later period this influence of lying on one or the other side became less marked and constant. But throughout the complaint, rest in the horizontal position had invariably the effect of diminishing the chylous condition of the urine. The morning urine, after the rest of sleep, was always the least milky; and that of the evening, after the fatigues of the day, the most so. The general health was only slightly affected. He was a little less capable of exertion, more easily fatigued, very sensitive to cold, and somewhat depressed in mind; there was also a dull pain in the left lumbar region. The exhaustion appeared to increase as the disease continued, but he was not compelled to suspend his somewhat arduous occupation for more than a day or two. He noted that a hemorrhoidal flux with which he had been previously affected, ceased entirely from the moment the urine became milky. In July and August the patient spent a month at the sea-baths of Warnemünde, where he led a very quiet life, but did not bathe. Here the urine suddenly became clear and normal, and continued so for a fortnight; but before he left, it became as suddenly, intensely milky again. At a still later period more frequent variations in the chylous and non-chylous condition of the urine were observed than in the beginning; but no intermission as long as that noted at Warnemünde occurred again. It often happened that amid a long series of chylous emissions a normal one would be suddenly interposed, and it was not always possible to find any cause for this sudden change in the circumstances of the patient.

It was observed that the skin was markedly less disposed to sweating than previously, and sometimes there was a disagreeable dryness of the cutaneous surface. The urine was notably increased in quantity, especially in the earlier periods, when it exceeded five pints in the twenty-four hours.

The examination of the urine yielded the following results: It was opaque, almost milk-white with a tinge of red, reaction acid, with a stale sweetish odor. It coagulated spontaneously, sometimes after standing a few minutes, sometimes after several hours. It never gelatinized within the urinary passages. It coagulated with heat and with nitric acid. When it was allowed to rest for eighteen hours in a glass, a thin, perfectly white layer gathered on the top, and a reddish deposit sank to the bottom. In the former a vast quantity of fat-molecules were found; in the latter, blood-corpuscles and small dark-red clumps of blood. Ether cleared the urine almost completely, and the extracted fat was solid at ordinary temperatures. When the urine was boiled, it passed through a filter perfectly clear, and possessing all the qualities of healthy urine —the fatty matter having been entirely retained on the filter by the coagulated albumen.

The following table exhibits the proportion of fat and albumen at different times of the day.

Table exhibiting the proportion of fat and albumen at different times of the day in Ackermann's case of chylous urine.

Date.	Hour of micturition	Antecedent condition	Specific gravity	Color.	Fat per cent	Albumen per cent.	Sediment.
Oct. 15	7 A. M.	Sleep.	1024	. . . .	0	0	None
" 16	11 A. M	2 hours' sitting.	1025	Yellowish-white.	0.82	0.506	Fibrinous flakes.
" 18	11 A. M.	2 hours' tuition out of the house.	1011	White, with little yellow.	0.34	0.145	A few pink fibrine flakes.
" 19	noon.	3 hours of the same.	1024	. . . . .	0 47	0.426	Shreds of fibrine.
" 20	5 P. M.	3 hours of the same.	1018	Light yellowish white.	0 26	0 968	Brownish-red clumps.
" 21	6 P. M.	3 hours of the same	1010	Light yellowish-white.	0.09	0.420	None.
" 23	10 P. M.	3 hours' sitting and half-hour reclining	1011	. . . .	0 24	0.202	None.
" 24	7 A. M.	Sleep.	1021	. . . . .	0 10	0 647	Shreds of fibrine.

Various remedies—among them gallic acid—were tried in this case with little or no evidence of success. In the beginning of December, 1859, he left off all medicines, finding them of no effect on his urine, and feeling his general health satisfactory. Toward the end of January, 1860, he found himself one evening, after a very heavy day, greatly exhausted, and chilly, and therefore went earlier than usual to bed. The urine on this evening was strongly chylous. Next morning, on the contrary, the urine was perfectly normal; the succeeding emissions were similarly healthy, and from that day forth the disorder did not return. For three years he has continued to pass perfectly natural urine. A few days after this favorable termination, the hemorrhoidal flux returned, and has continued since with its former frequency.[1] (Ackermann, "Deutsche Klinik," 1863, Nos. 23 and 24.)

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[1] This case came under the notice of Dr. Thudichum in 1864. The patient had returned to England, and the chylous state of the urine had reappeared, in consequence, as the patient believed, of higher living. Brit. Med. Journ., 1864, p. 611. Elaborate observations on a case of non-parasitic chyluria will be found in a paper by Siegmund, Berlin. Klin. Wochenschr., 1884, No. 10.

The general health of persons affected with chylous urine suffers in varying degrees. Some persons preserve their embonpoint undiminished; but the larger number are markedly emaciated. The patients generally complain of lassitude, incapacity for exertion, pains in the loins and the epigastrium. The unnatural drain of the nutritive material explains most of these symptoms. Sometimes there is an excessive appetite; more commonly the appetite is natural or indifferent. The long series of years, during which persons may void chylous urine, without serious impairment of their health, shows the comparative innocuousness of the complaint. In Quevenne's case (cited by Rayer) the patient, a native of the Isle of Bourbon, commenced to pass chylous urine at the age of 25. From this period to the age of 73 she constantly passed chylous urine. At 73 the urine became natural, and the patient thought herself cured; but after about fourteen months, the urine became again as chylous as ever, and continued so until she reached the age of 78, beyond which the report does not go. In Dr. Elliotson's case the disease lasted, off and on, for 28 years, without seriously affecting the health. When death has occurred in cases of chylous urine, it has been occasioned by some independent malady. In Dr. Priestley's case the kidneys presented the fatty form of Bright's disease, and the lungs were tuberculous. At his Gulstonian Lectures, in 1831, Dr. Prout exhibited the kidneys of a girl of 15 who had been passing chylous urine. She was said to have died of inflammation of the bowels; the kidneys were perfectly healthy. Dr. Isaacs had an opportunity of examining the body of a sailor, who during life had been in the habit of passing chylous urine, and who had died of general tuberculosis. The kidneys contained a few nodules of secondary tubercle, but were otherwise perfectly healthy.

Etiology.—Chylous urine prevails mostly in youth and middle age. Of 30 cases collected by me, 3 were under twenty; 7 between twenty and thirty; 11 between thirty and forty; 6 between forty and fifty; and 3 over fifty. The youngest example is mentioned by Prout, in a male infant of 18 months; the oldest is Quevenne's cases, in which the patient reached the age of 78 years.

Of these 30 cases, 19 were males and 11 females; but it appears, that in the countries where the disorder is endemic, it is more common among women than men. The greater frequency of it among men in the European cases is explained by the far greater number of men, who, as sailors, merchants, colonists, etc., pass a portion of their lives in tropical climates.

As to the predisposing causes of the disease, the only one made out with certainty is residence in certain tropical countries. Twenty-four cases (out of 30) were persons who had been born, or had passed a portion of their lives, in the Mauritius, Isle of Bourbon, Brazil, West Indies, or India.[1]

The best authenticated cases in persons who have never been out of Europe are—a case related by Prout; Mr. Gossett's case, cited by Bird; and Mr. Cubitt's case, cited by Beale; and the case recorded by myself.

The state of the blood has been examined by Guibourt, Bence Jones, and Ord. None of these observers found a milky state of the blood-serum. Guibourt (cited by Rayer) obtained nearly twice as much fat from the blood of a Brazilian affected with chylous urine as from healthy blood. Bence Jones, on the contrary, found in the blood of a person whose urine was milky both before and after the bleeding, no increased proportion of fat.

Pathology.—It has been generally assumed that the fat, albumen, and fibrine of chylous urine are derived from the blood, and pass into the urine through the kidneys. Dr. Prout says: "The proximate cause of this affection seems to lie partly in the assimilating organs, and partly in the kidneys. The chyle, from some derangement in the processes of assimilation, is not raised to the blood standard, and consequently being unfit for the future purposes of the economy, is, agreeably to a law of the economy, ejected through the kidneys; but these organs instead of disorganizing it, or reducing it to the crystallized state, as usual, permit it to pass through them unchanged."

But any view which supposes that the unnatural ingredients of chylous urine are derived from the blood circulating in the kidneys presents great difficulties. The rapid alternation of urine intensely chylous or lymphous, with perfectly natural urine (sometimes witnessed within intervals of an hour or two) seems incompatible with such a supposition. It is also incredible that blood, albumen, and fibrine should pass from the blood into the urine through the kidneys without being accompanied with casts of the uriniferous tubes. The absence of organic disease in the kidneys, and of any clearly made-out derangement in the composition of the blood, also militates against such a view.

I believe that the true pathology of chylous urine is to be sought for in the lymphatics of the urinary channels; and that the real analogues of the disease are to be found among those curious cases of chylous and lymphous discharges from the cutaneous surface, of which a number of examples have been published in late years.[1] It would appear, in our present state of knowledge, that some of these cases are due to a parasitic cause, and others to a structural change of a non-parasitic character. The following remarkable example may be taken as an illustration of the non-parasitic cases:

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[1] The endemic prevalence of chylous urine in these countries is thoroughly attested. The various speakers in the discussion at the Medical Society of Rio Janeiro, reported by Rayer, allude to the disorder as a common one among negroes. Dr. Prout states that Mr. Thomas, a practitioner from Barbadoes, informed him that he had seen at least a dozen well-marked cases in negroes in a practice of ten years. Dr. Carter also says that the more ordinary forms of chylous urine are "not uncommon" in Bombay. Dr. Bancroft, of Brisbane, Australia, writes me that cases of chylous urine are not unfrequent in the colony of Queensland.

W. Robinson, admitted into the Manchester Infirmary, September 21, 1868, æt. 45, always a resident in Lancashire, was troubled about two years before admission with a succession of large subcutaneous abscesses in various parts. One of these was situated on the lower part of the abdomen; it was a long time in healing, and one night he picked off the scab which covered the scar; and from beneath this exuded, during the night and the next day, a large quantity of fluid resembling gum-water. After this, he noticed a number of pale, transparent vesicles, no larger than pins' heads, scattered in the right iliac region over and around the site of the old abscess. When he first observed them they were ten or a dozen in number, but in a few weeks they began to spread and multiply, until in a few months, they dotted the surface of the lower part of the abdomen, almost as low as the pubes on the one hand, and as high as the umbilicus on the other. Some of them, also, began to discharge a pale watery fluid. By-and-by the vesicles and the discharge began to assume a thick, milky appearance, and gradually they assumed the condition in which they were found on his admission into the Infirmary.

On admission, the lower part of the abdomen was seen to be studded with numerous vesicles filled with a milk-white fluid. These were arranged partly in irregular groups and in part singly. Some of the groups contained three or four, others eight or twelve, vesicles, closely aggregated together. Some of the vesicles were so small that they were only just visible to the naked eye, others were as large as peas, and between these extremes were others of every intermediate size. Most of them were hemispherical, and some were oblong or irregular, as if two or more had coalesced. In the smaller ones the vesicular membrane appeared quite transparent, without a trace of organization, their opaque-white contents shining through them like drops of rich milk; but a few of the largest ones were distinctly marked by meandering lines of delicate bloodvessels, giving them a faint rose-color.

The skin over the affected area was thick and soft, and of a dull red color. When pressed with the fingers it yielded an almost spongy impression, but it did not pit. The integument was manifestly hypertrophied, and this gave to the lower part of the belly a protuberant appearance. This dull-red tumid area was somewhat more extensive than the limits of the vesicles, and faded at the circumference into the healthy skin about an inch beyond the furthest vesicle in all directions.

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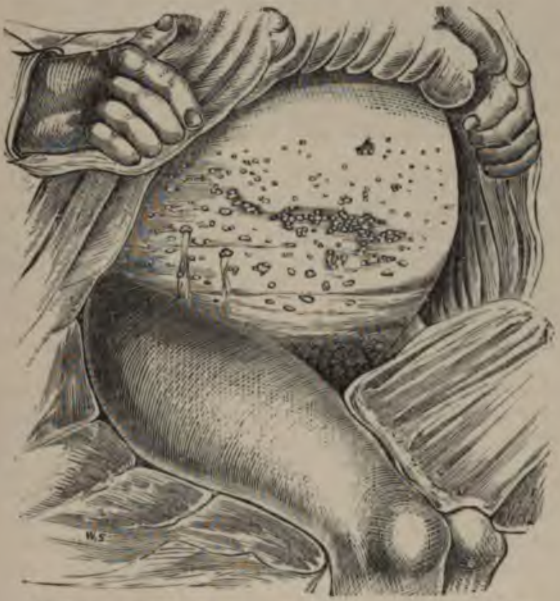
[1] Med.-Chir. Trans., vol. xlv. p. 189. Several cases of chylous discharge from the cutaneous surface (but without chylous urine) have been recorded. *See* A. B. Buchanan, Med.-Chir. Trans., xlvi. Fetzer, Archiv. f. Physiol. Heilk., vii. p. 128. Carter, Med.-Chir. Trans., xlv. Demarquay, Mém. de la Soc. de Chir., iii. p. 139. Also Trans. of Clin. Soc., vol. ii.

The skin around the larger vesicles, and groups of vesicles, was raised into soft nipple-like elevations, and had a more decidedly spongy feel than elsewhere. Slight pressure caused no pain, but the whole area was more or less tender on deep pressure.

In their normal state the vesicles were closed, and the immense majority remained throughout in this condition; but some dozen or so of the largest vesicles were at one time or other in a ruptured state, and discharged immense quantities of a chylous or lymphous fluid. The cause of rupture appeared to be some slight movement or violence. Sometimes the act of turning in bed sufficed to set the discharge in motion. It rarely happened that more than two or three vesicles were discharging at the same time. The quantity of this discharge, and its occurrence and arrest, were most irregular. Sometimes several pints would be discharged in a day and night, and sometimes only sufficient to moisten the cloths with which the patient girded himself. The patient was sometimes continuously wet for three or four weeks; at other times the flow would continue only a few hours or a few days. The intervals of complete dryness were similarly uncertain, and varied from a day or a few days to two or three weeks.

FIG. 48.



Case of W. Robinson—appearance of the eruption on the abdomen.

The character of the discharge also varied; sometimes it was like thick milk, sometimes like skimmed milk, and sometimes perfectly pale, like gum-water. Whether white or pale, it was always spontaneously coagulable, and white or yellowish clots collected about the seat of discharge. The color of the unruptured vesicles varied in correspondence with that of the discharge, from milk-white, or opalescence, to pale straw. The degree of milkiness at any particular moment was always the same in all the separate vesicles, showing that the cause of variation was not a local one particular to any vesicle, but something affecting the eruption generally, and depending presumably on the state of the blood.

The vesicles varied not only in color, but also in fulness and turgidity; and it was noticed that the whiter they were the more distended they appeared, and that when they were pale they were also more flaccid.

Two circumstances affected, though somewhat irregularly, the whiteness and fulness of the vesicles, namely, the general state of the patient's health, and the digestion and assimilation of food. On the days when the patient was out of sorts or feverish, the vesicles were paler and more flaccid; but when the appetite and sleep returned, the vesicles became milky and turgid.

The effect of food was found to be tolerably constant in kind, though not uniform in degree. The vesicles were paler in the morning before breakfast, after the prolonged fast of the night. At this period they were often quite lymphous. Soon after breakfast they began to grow fuller and whiter, and, as a rule, the milkiness increased through the day, attaining its maximum some seven or eight hours after dinner. Of course, the appearance of the discharge, if there were any, followed the same rule.

The vesicles seemed to be situated in the substance of the cutis, and their surface-wall was evidently composed of something besides epithelium. In the larger vesicles their base was raised, and consisted of soft cutaneous tissue; and capillary vessels could be seen travelling over their transparent summits. When a vesicle was gently pressed with the tip of the finger, it was immediately emptied, its fluid contents escaping into the deeper parts. After the pressure was withdrawn the vesicle slowly filled again. There was no direct communication between neighboring vesicles, and when one was ruptured and discharging, the vesicles around it still appeared full and turgid. It was noticed, however, that when the discharge had been very free for some hours, all the vesicles appeared flaccid. Even when a whole cluster was compressed, the neighboring vesicles did not appear more distended. The idea conveyed by the study of the effects of pressure on different vesicles and groups of vesicles was, that each vesicle communicated with a more deeply situated reservoir of anastomosing channels. When a vesicle was pricked, the flow from it immediately began, and it continued at a steady rate for hours together. On one occasion the rate of flow from a punctured vesicle was tested, and found to be equivalent to eight ounces per hour.

The characters of the discharge, whether it was milky or opalescent, were always essentially the same. After standing a few minutes it set into a tremulous jelly. In a few hours there was a separation into clot and serum. It coagulated with heat and with nitric acid, but not with acetic acid. When shaken with an equal bulk of ether the white appearance was removed, and the fluid became transparent and yellowish like blood-serum. These reactions prove that it contained fibrin, albumen, and fat, and that it differed essentially from true milk in not containing casein. The reaction was always alkaline. The varying degree of milkiness was, of course, due to the varying quantity of fatty matter. Under the microscope, myriads of minute fat molecules were seen, sometimes mixed with larger oil globules; in addition to these, pale corpuscles, identical in structure with the white corpuscles of the blood or chyle, were always present, but not in large numbers. No other organic forms were ever seen except the transparent fibrillæ of coagulated fibrin.

The fluid is thus seen to be similar in character to chyle when milky, and to lymph or liquor sanguinis when pale. It is also identical with the admixture which takes place in cases of chylous urine. A case of chylous urine[1] happened to be in the Infirmary at the very time the present case was under observation, and neither chemically (excepting proper urinary ingredients) nor microscopically could any distinction be made between them. Still more significant of this alliance was the fact to be noted presently, that on two separate days this man did actually pass chylous urine.

The condition of the *urine* was carefully noted during the progress of the case. It was generally found to be remarkably scanty in quantity and of high specific gravity. When the discharge from the vesicles was abundant, the quantity of urine ranged from 13 to 18 ounces in the twenty-four hours. When the eruption was dry, the urine was somewhat more abundant, and varied from 18 to 25 ounces—on one occasion it reached 34 ounces, and on another 40 ounces, which was the largest flow chronicled during his long sojourn in the Infirmary. The specific gravity varied from 1025 to 1032; it frequently deposited lithates, but did not contain either albumen or sugar. The scantiness of the urine was partly due to the voluntary abstention of the patient from drink. He believed that drinking always increased the flow of the discharge; and he endured constant and severe thirst in order to check this loss.

On December 2d the urine was voided milky on two occasions. It presented all the ordinary characters of chylous urine. Again, on January 15th, the patient passed three ounces of chylous urine, and on the following day fat was found in the urine with the microscope, though not in sufficient quantity to produce a milkiness of the secretion. During these two days the eruption was dry. With these exceptions the urine continued of normal composition throughout, and free from albumen.

The only general symptoms referable to the disease on the abdomen, and the discharge, were attacks of chilliness and shivering, with a sense of great weakness. These occurred repeatedly when the discharge was copious and long continued. He also complained occasionally of aching pains in the abdomen and of indifferent sleep; but, as a rule, he was in a state of fairly comfortable health until tuberculous symptoms broke out.

When the patient entered the Infirmary in September, he did not appear to be affected with any disease except that on the abdomen. But in March of the succeeding year he began to lose weight, and the temperature of the body, which previously had ranged from 97° to 98.6°, began to rise, and ranged from 99° to 102°. The physical signs and symptoms of pulmonary phthisis also made their appearance, and steadily progressed. His health slowly declined, until at length he was unable to leave his bed.

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[1] The case already reported at p. 338.

The eruption withered slowly as the pulmonary disease advanced; the vesicles became persistently pale and flaccid; the discharge became watery and scanty, and finally ceased some five days before death. The state of the eruption the day before death is thus described by my clinical clerk, Mr. Patchett. "The vesicles have lost their character of vesicles altogether, they seem converted into small furfuraceous scales of different colors, some being of a reddish-yellow, others of a raspberry, color; the small vesicles scattered over each flank look exactly like flea-bites."

The urine was reduced to six and eight ounces per day in the last week, and the symptoms assumed the so-called typhoid character—with low muttering delirium, indifference, picking at the bedclothes, and finally coma. Death occurred on May 22d.

Autopsy Twenty-one Hours after Death.—Both lungs were studded with gray granulations intermixed with larger masses of gray and yellow tubercle, some of which were softened. Two small vomicæ were found in the left apex, and one in the right. Tuberculous ulcers were also found in the small and large intestines. The bronchial and mesenteric glands were enlarged. The liver weighed sixty-four ounces, and the spleen nine ounces; both organs were healthy. The kidneys and bladder were healthy. The integument of the hypogastrium was much thickened and spongy, contrasting strongly with the emaciated integument over the thorax. The lining membrane of the bladder was minutely examined, and appeared smooth, glistening, and healthy throughout. No enlargement or unnatural condition of the thoracic duct or of the lymphatic vessels or glands could be detected. A considerable piece of the abdominal wall, embracing a portion involved by the disease and a portion extending beyond into the healthy skin, was cut out for further examination.

Examination of the Skin in the Diseased Area.—On making a vertical section through the skin and subjacent parts, it was at once perceived that the disease involved essentially the cutis vera and the subcutaneous tissue. The tendinous, muscular, and peritoneal strata were in every respect perfectly normal. The skin was immensely thickened, and formed, with the subcutaneous tissue, to which it was structurally united, a thick pad or layer of tissue varying from half an inch to an inch thick. When fresh, the cut surface had a pale rose and somewhat fleshy or granular appearance. This tissue was traversed by short channels or lacunæ, varying from the width of a crow-quill to that of a hair. By making numerous thin sections vertically and horizontally and examining them with a lens and a microscope, these lacunæ could be seen to communicate freely with each other by small smooth orifices. The vesicles evidently constituted the surface boundaries of the more superficial lacunæ. The lining membrane of the lacunæ and of the vesicles was smooth and glistening; and, when gently scraped with a knife, it yielded a small quantity of a whitish débris, which, under the microscope, resolved itself into spheroidal and nucleated cells resembling those which were found in the discharge during life.

Here are evidently the elements of a glandular structure—a membrane lined with spherical nucleated cells. But the analogy is rather with the ductless follicles of Peyer's patches, and still more with the ganglia of the lymphatic chains than with the glands engaged in the regular work of secretion and possessing excretory ducts. The new structure had no connection with the normal glands of the skin. The funnel-shaped orifices of the sweat-glands could be seen opening independently on the surface in the hollows between the vesicles, and the hair follicles presented their normal appearance.

The chief interest in the case lay in the light which it throws on the pathology of chylous urine. It can scarcely be doubted that the case was generically identical with that curious disorder. The absolute similarity of the discharge with the fibro-albuminous and fatty elements added to the urine in chylous urine, the sudden appearance and cessation of the discharge, the capricious terms of the duration of the discharge in the two disorders, and the actual occurrence of chylous urine on two occasions in the case of Robinson, scarcely leave any room for doubt on this point. Had the disease in this case, instead of occupying the subcutaneous tissue of the abdomen, been developed in the submucous tissue of any part of the urinary passages, it is evident that the conditions for the production of an ordinary case of chylous urine would have existed. It is even almost certain that some small part of the urinary membrane—probably that of the front of the bladder—was actually invaded by the disease which affected the abdomen, but no anatomical traces of such extension could be detected at the autopsy, owing probably to the fact that in the last few weeks of life the morbid process had retrograded, and had consequently left no appreciable marks on the surface of the bladder.

It may be assumed with some confidence, that in some cases of chylous urine the disease is essentially of the same pathological nature as the disease on the abdomen of Robinson. And looking to the absolute identity of the discharge in these cases with chyle and lymph, it is difficult to avoid the impression that the structures which produce this discharge are anatomically analogous to the lacteal and lymphatic tissues. The examination of the skin of the abdomen in Robinson's case gave strong support to this view. When the preparation was fresh, the thick, soft layer, into which the skin and subcutaneous tissue were converted, had very much the pale flesh-color and general appearance of lymphatic gland tissue; and the short communicating lacunæ traversing it in all directions, suggested a structure not dissimilar to an immense exaggeration of the lymphatic plexus. I found it impossible to resist the idea that this was really the true pathological solution of the case, and that a similar solution applied to cases of chylous urine.

It is well known that the skin with the subcutaneous tissue, and the mucous membranes with the submucous tissue, are exceedingly rich in lymphatics, which form a close network of communicating channels in these situations. It is further known that the cells lining the lymphatic channels, especially those of the lymphatic glands, perform a glandular function, and impress important changes on the lymph passing through those channels.

Now let it be supposed that at some spot the lymphatic network becomes immensely hypertrophied: that its channels become varicose (as it were): that the contained cells assume by degrees the property and function of the cells lining the lacteal ducts and lacteal glands: that the more superficial of these varicose enlargements project above the surface of the skin or of the urinary mucous membrane, as the case may be; and, lastly, let some of these superficial enlargements become ruptured and discharge their contents externally or into the urinary passages, and the conditions are presented for the production of chylous urine, or of such a case as that of Robinson and others of a similar class to which reference has been made (*see* p. 346, footnote).

It is always satisfactory in studying any rare disease, to be able to refer it by analogy to some preëxisting well-known category, and the view just presented of the pathology of chylous urine and the allied disorder of the skin, finds its exact analogy in those hypertrophies of the bloodvessels which constitute venous nævi, erectile tumors, and aneurisms by anastomosis—all of which are exaggerations of hypertrophies of the normal arterial or venous plexuses.

It rarely or never happens that any tissue suffers morbid hypertrophy without some degree of modification of its normal structure; and the hypertrophied lymphatic tissue which I have suggested as one cause of chylous urine and the allied condition in the skin, is undoubtedly modified by the morbid impulses which generate it. Not only is the anatomical structure considerably altered from the normal type of lymphatic tissue, but the function of the cells also suffers a modification. The cells which, in the normal state, elaborate lymph, in the morbid state come to produce chyle, or a fluid intermediate between lymph and chyle. These modifications are, however, strictly within the limits which we generally find in other morbid hypertrophies.

A new and important light has been thrown on the pathology of chyluria and those cases of cutaneous chylous discharges which occur endemically in India, by the researches of Dr. T. R. Lewis. Dr. Lewis discovered that the blood and urine of patients suffering from chylous urine contained numbers of actively moving microscopic worms, to which he gave the name of *Filaria sanguinis hominis*. These have since been shown to be the embryos of an adult worm, which was not discovered until some time afterwards. The reader is referred to Chap. XIII., Section III., for a description of the parasite and the other symptoms which attend its presence in the body. In a post-mortem examination of a case of chyluria, Dr. Lewis found in the kidneys vast numbers of filariæ, both in the cortical and pyramidal portions. There were also found "numerous translucent oil-like tubules of a somewhat varicose appearance running alongside the uriniferous tubes as if the lymphatics or minute bloodvessels of the part had been plugged." The organs did not appear otherwise diseased.

My former pupil, Dr. Bancroft, now practising in Brisbane, Queensland, Australia, informs me that chyluria is not uncommon in that colony. He has detected filariæ both in the urine and in the blood of patients so affected, and has also discovered the adult worm which gives rise to them.

It may be supposed that aggregations of these little animals in the kidneys, or some other part of the urinary tract, give rise to rupture of the lymphatics and a leakage of their contents into the urinary channels, and in this way produce chyluria. A further account of the life history of the parasite will be found in Chapter XIII., to which the reader is referred.

The following abstract of Dr. Mackenzie's case will serve to illustrate the course of chyluria due to filarial embryos, and is specially interesting from the completeness of the observation, and from the light which it throws upon the relations of the filarial embryos to the periods of the disease:

F. H. C., aged twenty-six, was born at Madras, and lived in India up to six months of coming under the care of Dr. Mackenzie. About one month after arriving in England he noticed that his urine was increased in quantity, that it contained clots, and appeared slimy. In about a week the urine gradually assumed a milky appearance, and shortly became as "white as milk." On May 6, 1881, whilst lying down in the middle of the day he was seized with a violent pain extending from the left loin to the left testicle. The pain lasted for about twenty-one hours, and during its continuance the urine was of a deep blood color. He gradually lost flesh, the urine became more copious, and formed on standing gelatinous clots. The patient was admitted into the London Hospital, under Dr. Mackenzie, on August 11th.

He was then a well-made, healthy-looking man. His appetite was good, but he slept badly, having to rise frequently to pass urine. No disease of the chest or abdomen could be detected. His weight was 9 st. 1 lb., while on arriving in England it was 11 st. 8 lbs. The urine resembled rich milk mixed with a little blood. A few minutes after being passed it coagulated to a soft tremulous jelly, the coagulum afterwards breaking down. When freshly passed, the urine had a sweet odor, but on standing became fetid. The quantity passed daily was on the average 120 ounces. It had a specific gravity of about 1010, was faintly alkaline or neutral, and contained no sugar. It contained albumen. When shaken with ether, the urine lost its milky character. The day urine had a brownish tint, almost completely coagulated, and contained much blood, while the night urine was more milky, did not form so large a coagulum, and contained less blood. Filariæ were found in the urine both dead and alive. The blood contained large numbers of embryo filariæ when examined at night time. Between 9 A. M. and 6 P. M. no filariæ were found; at 6 P. M. a few were seen; these had increased in number at 9 P. M., were in the greatest number at midnight, and gradually decreased in number up to 9 A. M., when they had entirely disappeared. At night it was computed that from thirty-six to forty millions of filarial embryos were present in the blood.

The hours of meals were altered to four hours later without any effect on the filariæ, but the day and night urines were rendered almost alike Complete reversion of the hours of movement and rest, and corresponding change in the meal times, caused similar reversion in the filarial periodicity, the maximum number in the blood being then found at noon, and few or none at midnight.

Under treatment the patient's health improved and his weight increased. On October 21st, after exposure to cold, he had a severe rigor with vomiting, headache, and pain in the epigastrium and right hypochondrium. Temperature 104°. The patient coughed without expectoration, and had a pain in the right side, increased on coughing. On October 23d the left shoulder was tender and swollen. Later on, a swelling appeared just above the left clavicle, the skin over which was red. This increased in size, gradually assumed the characters of an abscess, and was opened antiseptically on November 4th, pus and blood escaping. Double pleurisy afterwards developed itself, and further collections of pus formed in the left arm. The patient gradually lost strength, and died on January 10, 1882. From October 22d no filariæ were found in the blood, and while they were seen as usual on October 20th, on the 21st they were very feeble and soon died.

At the autopsy there was found empyema of the right side, and on the left side pleurisy. The right lung was collapsed, and the left œdematous. The kidneys showed early suppurative nephritis and several wedge-shaped patches were found in the cortex. The thoracic duct commenced in a dense mass of dilated lymph sinuses extending from the bifurcation of the aorta below to the aortic opening of the diaphragm above. The thoracic duct was pervious for 1½ inch above the diaphragm, then filled with loose clot for a similar distance, after which it was lost in a tough, thick mass which was apparently of inflammatory origin. The iliac, lumbar, and renal lymphatics were dilated, but especially those in the left renal region.

It was supposed that the parent worm had become dislodged in the rigor, and becoming impacted in the thoracic duct, had excited inflammation, in the midst of which it perished.

Treatment.—Hitherto the treatment of this disorder has proved very unsatisfactory. It generally persists in spite of every remedy, or disappears without any. The physicians of Rio chiefly recommend salt-water baths, and iron internally. Mineral and vegetable astringents have been tried repeatedly with small evidence of success. The best results have followed large doses of gallic acid. Dr. Waters and Dr. Bence Jones gave from one to two drachms a day.

Dr. Bunyan, of George Town, British Guiana ("Lancet," 1846, I. 95), relates a very interesting case, in which the disease had lasted ten months. Various remedies were tried without success. On the advice of an old negress, the patient took a decoction of mangrove bark (Rhizophora racemosa), in ounce doses, four times a day. In seven days, he was so greatly improved that he discontinued the medicine for two days, when the symptoms returned. The medicine was resumed in increased quantity, and continued for several days, until all the symptoms had entirely disappeared. Afterwards he suffered two returns of his disorder, which were immediately cut short by the use of the mangrove bark. I know not whether the mangrove bark has anti-parasitic properties, but if it has its success in the treatment of chyluria is comprehensible. It would certainly be worth a trial to treat cases of chyluria, in which filariæ are found in the blood, by anti-parasitic remedies, and especially by large and sustained doses of the iodide of potassium.

The effect of diet was investigated by Dr. Bence Jones. He found that the urine was somewhat less chylous with vegetable than with animal food; he also found that the pressure of a tight belt round the loins relieved the pains in the lumbar regions, and seemed to improve the condition of the urine a little.

Dr. Dickinson, in his case, believing that the chyluria was due to regurgitation of chyle from the thoracic duct into the bladder, applied the abdominal tourniquet, with a marked amelioration of the condition of the urine. The effect of individual applications of the tourniquet diminished after a time, but a slight permanent improvement was observed. The chyluria finally ceased after the use of injections of perchloride of iron into the bladder.

PART III.

ORGANIC DISEASES OF THE KIDNEYS.

CHAPTER I.

CONGESTION OF THE KIDNEYS.

Under the title of Congestion of the Kidneys, I propose to consider those less serious, and for the most part secondary, renal derangements which are occasioned either by an undue determination of blood to the organs (active congestion), or some mechanical obstruction to the return of blood from the organs (passive congestion).

Renal congestion, both active and passive, if sufficiently intense, is attended by the presence of albumen in the urine (generally in small quantity), sometimes with blood, and casts of the uriniferous tubes. Dropsy is not a symptom proper to renal congestion; when present it depends on other causes, commonly heart or lung disease.

Active congestion is produced by—overdoses of certain irritants (cantharides, turpentine, etc.); by exposure to cold; it is a common instance in all febrile and inflammatory complaints; it occurs in saccharine diabetes; probably in some cases of hypertrophy of the left ventricle; and it is found also in the opposite kidney when one kidney has become disabled.

Passive congestion accompanies—regurgitant heart disease; obstructions in the lungs (emphysema, pleuritic effusion); pressure on the emulgent veins or inferior cava (pregnancy, abdominal tumors).

If the determining cause of the congestion be a persistent one—as in valvular heart disease or diabetes, organic changes are at length produced in the kidneys, which bear a strong resemblance to, if they are not identical with, certain forms of Bright's disease.

Accordingly, several of the conditions here considered have been arranged by other writers (Johnson, Frerichs, Griesinger, Bamberger, Wagner) among the varieties of Bright's disease. But although there are unquestionable affinities between the two classes of cases, there are also differences so marked, in their symptoms, progress, and general clinical history, that it only tends to confusion to unite them under one heading.

It will greatly facilitate our comprehension of the relations subsisting between certain changes in the composition of the urine, and certain disturbances of the renal circulation, if we take a review of the experimental researches which have been made in this direction.

Mr. George Robinson was the first to demonstrate, that a complete or partial impediment to the return of blood by the renal veins caused albumen, blood, and sometimes fibrin to appear in the urine. He operated solely on rabbits. In one set of experiments, he placed a tight ligature round the renal vein: in a second set, the obstruction was made incomplete—a certain amount of blood being still permitted to circulate through the kidneys. In both these sets of experiments the urine invariably became more or less albuminous, and in most cases bloody. The kidney, of which the vein had been thus obstructed, was in every instance found heavier than its uninjured fellow. The proportion between them varied from 1½ : 1 to 3 : 1.

Frerichs repeated these experiments on dogs, rabbits, a cat, and a frog, with identical results. In four out of ten experiments, he also detected casts of tubes in the urine, and in one, renal epithelium. Weissgerber and Perls, Posner, and Germont[1] have repeated these experiments and have obtained similar results.

The experiments admit of easy explanation. The blood accumulates behind the impediment, and causes an increased lateral pressure upon the walls of the renal vein and its branches. This tension is transmitted backward to the renal capillaries, which are thereby distended, and their walls attenuated, creating a condition highly favorable to the transudation of the serous constituents of the blood through their coats. If the tension be sufficiently great, blood corpuscles escape from the vessels either by diapedesis or actual rupture, and pass with the albumen into the urine. It is probable that these consequences take effect earliest in the tubules of the medullary portion of the kidney, for with these the branches of the renal vein come into close relation; thence the pressure is transmitted through the renal capillaries to the Malpighian clusters, where there exist anatomical facilities for ready passage of blood into the urine.[2] Cohnheim is of opinion that it is not the mere increase in blood-pressure which causes albumen to transude, but a coincident alteration of the epithelium covering the glomerulus.

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[1] Thèse De Paris, 1882.

[2] *See* Senator, Die Albuminurie, 1882.

Increased pressure in the *arterial* system does not so easily cause albumen and blood to appear in the urine. Robinson sought to test the effect of increased arterial pressure on the composition of the urine, by directing a stronger stream of blood than natural into the kidneys. First he removed one kidney, thinking that the physiological determination to the other might suffice to cause albuminuria. The experiment was repeated five times, and only in one instance did the urine become albuminous. He then removed one of the kidneys and tied the abdominal aorta below the origin of the renal arteries.[1] In this way the utmost impulsion of blood into the remaining kidney was obtained, and both blood and albumen invariably made their appearance in the urine. His seventh experiment is a fair sample of his results.

Expt. 7. The left kidney of a middle-sized rabbit was removed, and weighed 54 grains. The aorta was then tied below the origin of the renal arteries. The animal was killed at the end of two hours. The right kidney weighed 85 grains; it contained six or seven ecchymoses of various extent. The bladder contained about a drachm of urine, which was bloody and albuminous (loc. cit., p. 79).

These results have been confirmed by Frerichs and Meyer.

In the experiments of Hermann and Overbeck, another method of inducing artificial albuminuria is pointed out. Hermann's method consisted in tying up the renal arteries for a short time, and then removing the ligature. The urine which was secreted after the reëstablishment of the circulation was always found albuminous. Overbeck interrupted the circulation in other ways. In one set of experiments, he blew up a bladder previously introduced empty into the heart; in the second set, asphyxia (and consequent arrest of the blood-current) was produced by compressing the trachea. In the former case the obstruction was maintained for about a minute, and in the latter for four minutes. In both classes of experiments, the urine which first flowed after the renewal of the circulation was invariably albuminous, and often bloody. The albuminuria thus provoked, generally lasted a few hours, and then passed away. When desquamation of the renal epithelium occurred, it always *followed* the appearance of the albumen. It could not therefore be the cause of it, as Johnson surmised to be the case in the albuminuria of Bright's disease.

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[1] Tying the abdominal aorta without removing one of the kidneys was performed twice by Robinson on weak animals; in one only did albumen appear in the urine. Frerichs states that he could only find traces of albumen in a few cases after such an operation. Meyer, on the other hand, saw abundant albuminuria follow this operation.

To explain the results obtained by Hermann and Overbeck, it may be supposed that the temporary stoppage of the blood-current created an obstacle in the renal capillaries—probably an accumulation of blood-corpuscles in the Malpighian tufts—which, when the circulation was restored, operated to raise the pressure in the minute arteries; in other words, it produced active congestion of sufficient intensity to cause albumen and blood to appear in the urine.[1]

My purpose in calling attention to these researches is to show, that simple hyperæmia or congestion of the kidneys (without inflammation), either from increased impulsion of blood into the kidneys, or from obstruction to the return of blood from the kidneys, is sufficient to determine the appearance of albumen and blood and even fibrinous casts in the urine.

An impeded circulation through the kidneys cannot, however, long persist, without inducing serious and permanent structural changes in the organs. The presence of blood-corpuscles, and fibrinous plugs, in the delicate tubular structures, must at length occasion more or less extensive destruction of these structures; and the continued hyperæmia must derange the nutrition of the glandular elements. How far these changes are of an inflammatory nature, cannot be precisely indicated. One of the most important results of a long continuance of this state of things appears to be, an excessive production of adventitious connective tissue, which eventually passes on to contraction and atrophy. To call these changes, "nephritis," is to use a term, which, to say the least, is calculated to mislead.

ACTIVE CONGESTION.

(*Catarrhal Nephritis of Virchow.*)

In the course of eruptive and continued fevers, of croup, diphtheria, cholera, erysipelas, pyæmia, acute rheumatism, pneumonia, and other inflammatory diseases, the kidneys partake in the general hyperæmia of the internal organs. Not unfrequently, however, they are the seat of a disproportionate determination of blood, and albumen appears in the urine. Generally speaking, the amount of albumen, in such cases, is a mere trace, but sometimes it is more abundant, and accompanied with a few blood-corpuscles, transparent casts of tubes, and scattered renal epithelium. There may be, at the same time, some tenderness in the loins. As soon as defervescence commences, the albumen diminishes, and in a few days vanishes altogether.

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[1] For other possible explanations, *see* p. 203.

The pathological state here described differs from genuine Bright's disease, which may likewise arise in connection with the same febrile maladies, in the absence of anasarca, in the undiminished excretion of urea, and in the period of its invasion. Albuminuria from congestion coincides with the acme of the pyrexia, and subsides therewith. Genuine Bright's disease, on the contrary, shows itself as a sequela, toward the close of the pyrexial stage or the commencement of convalescence.

An examination of the kidneys of persons who have died from the primary fever while laboring under renal congestion, reveals an enlarged and engorged condition of the organs, with minute ecchymoses on the surface, and great engorgement of the stellate veins. A large amount of blood flows from the cut surface of the kidney, and the glomeruli and accompanying vessels stand out as red points and streaks. On microscopic examination, the vessels of the glomeruli and the intertubular capillaries are swollen and gorged with blood-corpuscles, while numerous blood-corpuscles are seen in the lumen of the tubules and between the glomerular tuft and its capsule; the epithelial cells are swollen, very granular, sometimes show signs of fatty changes, and are frequently detached.

The frequency of this complication in zymotic diseases, varies in different epidemics. Rosenstein states that in a severe typhus epidemic, witnessed by him in 1857, the majority of the patients had transient albuminuria, with casts of tubes, and yet no serious consequences followed therefrom. In the sporadic typhoid of this city, albuminuria is decidedly rare.

Active renal congestion of a catarrhal nature, may also arise independently of any specific fever, simply from exposure to cold. Such cases are not very common, or perhaps, as Rosenstein suggests, they are often overlooked. The symptoms resemble those of a simple febricula, and, unless the urine chance to be examined, the disorder will probably be passed over as such. The following example is from Rosenstein:

A. B., æt. 39, previously healthy, experienced on the afternoon of the 7th of October a chill, followed by heat and severe pains in the renal region, which were accompanied with vomiting. When seen she was in a high fever, pulse 120, very thirsty, and without appetite. The urine was scanty, acid, albuminous; after standing, it deposited a sediment composed of uric acid, blood-corpuscles, epithelial casts, and free epithelium. Pressure on the renal region caused pain. She was cupped on the loins. On the following day the urine measured 27 ounces, specific gravity 1026, otherwise as before reported. On the 16th, the pulse was 92, skin moist and perspiring, general condition good. Urine in twenty-four hours, 28 ounces, specific gravity 1025, acid, free from albumen, containing only a few casts. On the succeeding days increased diuresis in, with diminished specific gravity of the urine. The urine continued free from albumen and formed elements (p. 98).

Cases of a similar nature connected with subacute rheumatism are not very uncommon. The following, which I saw with the late Mr. Mellor, may serve as an example:

The patient was a young lady of 26, who had been subject, for several years, to frequently recurring attacks of subacute articular rheumatism, which kept her in a continuously weak state of health. On April 14th, she took cold through walking in the wet, and was seized with tonsillitis. As this subsided, the urine was noticed to be bloody and to contain albumen. On the 15th of May I saw her for the first time. She was very pale and thin; there was considerable fever, pulse 108, the loins were painful and very tender on pressure, skin dry, with a tendency to frequent vomiting. Micturition was very frequent (20 times a day); the urine amounted to three pints in the twenty-four hours, specific gravity 1010; it contained a good deal of blood and albumen, and deposited uric acid very abundantly. The copious deposit which subsided when the urine was left in repose, contained numerous large transparent casts (some studded with epithelium) and much free renal epithelium. Neither casts nor epithelium showed any signs of fatty degeneration. There was also found a large number of pyelitic cells. Not a particle of dropsy or anasarca existed in any part.

The patient was dry-cupped over the loins, after which hot poultices were directed to be kept (frequently renewed) to the same region; a compound jalap powder was administered, and a citrate of potash mixture.

In four days the fever subsided, the pains disappeared, and the skin became moist. At the same time, the urine was far less frequently passed, and it contained much less blood, albumen, and casts. It still continued abundant in quantity, and deposited uric acid very copiously.

In the course of four weeks, convalescence was so decidedly established, that the patient was allowed to sit up. The albumen now scarcely exceeded "a haze" with nitric acid. She was put upon a phosphoric acid mixture, combined with phosphate of iron.

On June 24th the patient suffered a relapse. She was again confined to bed, and the previous treatment put in force. In a few days the feverish symptoms passed off; but a good deal of blood, albumen, and renal derivatives continued to be discharged. She was now put upon gradually increasing doses of dilute sulphuric acid, with most excellent effect. The urine steadily resumed its natural characters, and the patient's appetite and strength began to return.

On the 24th of July the urine had become free from albumen and blood, and convalescence was thoroughly established.

The case was, from the beginning, regarded as distinct from genuine Bright's disease, and considered as presenting the features of a catarrhal (rheumatic?) condition of the pyramidal parts of the kidneys, combined with some degree of subacute pyelitis. The total absence of anasarca, and the general portraiture of the complaint, forbade the idea of acute Bright's disease; while the state of the urine and the progress of the case appeared inconsistent with the chronic forms of that formidable disorder.

Certain irritants—cantharides, turpentine, cubebs, copaiba, nitrate of potash, and carbolic acid[1]—act as special stimuli of the urinary organs; and excite, when administered in excessive doses, hemorrhage from the kidneys and the lower urinary passages. Johnson relates an instance in which half an ounce of turpentine was taken for the expulsion of tape-worm. In a few hours the urine was bloody, and in the deposit "blood-casts" were discovered, together with a few small inflammatory cells, but no epithelium. Six days after, the urine contained less blood and albumen. The casts of tubes were still visible, and contained, besides the blood-corpuscles, a large proportion of inflammatory cells about twice the size of the blood-corpuscles.[2] The patient continued to pass more or less blood for some days longer. On the sixteenth day the urine was free from albumen and blood.

Bouillaud examined the effects of cantharides acting through the skin. He states that, almost constantly, when large blisters were applied to scarified portions of the skin, albumen appeared in the urine. After death, he found the mucous membrane of the pelvis and ureters, in other cases that of the bladder, injected, and covered here and there with false membranes. The kidneys were generally strongly congested and studded with minute ecchymoses. Albuminuria after cantharides usually disappeared in two or three days; in a few cases it lasted four weeks.

Two cases of poisoning by sulphuric acid are related by Leyden and Munk, in which albumen and casts appeared in the urine.[3]

Cases have also been described in which the external application of tincture of iodine, of styrax, and of petroleum, have given rise to albuminuria.

Frerichs enumerates irritants of this class among the exciting causes of genuine Bright's disease; and brings forward two cases by Reinhardt, in which abuse of copaiba and cubebs was followed by renal degeneration, which in one of them proved fatal. These cases are, however, as Rosenstein points out, inconclusive, because it is probable that the kidneys were already diseased before the use of the irritants was commenced.

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[1] Quinine in very rare cases produces congestive hæmaturia. This effect appears to be due to an idiosyncrasy. I have known one such person. Two cases are also reported in the Brit. Med Journ. for January, 1870.

[2] Were not these renal epithelia?

[3] Archiv f. path. Anat, Bd. xxii. S. 237. Hydrochloric acid also caused albumen, casts, and blood to appear in the urine, in a case reported by Gehle, Berlin. klin. Wochenschr., 1884, No. 22.

More recent experiments have shown, that genuine organic change in the kidneys may be produced by the internal administration of irritants; although the exact nature of the change is a matter of dispute amongst the several observers. Cornil,[1] by giving cantharides to animals, produced marked alterations in the epithelial cells, such as are found in parenchymatous nephritis. The cells were swollen, completely filled the lumen of the tubules, and contained fat granules and occasionally red blood-corpuscles. In many cells, too, globules of a hyaline material were seen. Similar epithelial changes were found by Browicz.[2] Dunin[3] describes changes in the cells, due to "coagulative necrosis." Aufrecht,[4] on the other hand, found interstitial nephritis, and succeeded in producing even a granular kidney. It is probable that the nature of the change depends on the dose of the poison and the method of its administration, small doses producing mere congestion, larger doses the epithelial changes, while small doses given repeatedly cause an overgrowth of interstitial tissue.

In a previous section it has been mentioned, that in the later periods of diabetes, albumen not unfrequently appears in the urine. The excessive action of the kidneys in this disease, keeps up a constant congestion of the organs: and, in the course of time, permanent anatomical changes follow—degeneration of the epithelium, increase of interstitial tissue, development of cysts, and other structural alterations, which are sometimes classified with genuine Bright's disease.

There is yet one other condition which seems capable, in rare instances, of producing an active congestion of the kidneys, sufficiently intense to determine albuminuria. In the compensatory hypertrophy of the left ventricle, which follows aortic regurgitant disease, the propulsion of blood into the aorta (when the orifice is patulous) takes place with very great force; and the tension of the arterial system at the close of the ventricular systole, rises considerably above the normal maximum, as is indicated by the full resistant pulse.[5] Practically, however, albuminuria traceable to hypertrophy of this kind, is rare. I have repeatedly examined the urine of persons with immense enlargement of the left heart, without finding albumen in more than three or four instances. The following is one of these, in which no tenable explanation of the albuminuria could be found except renal congestion from excessive power of the left ventricle.

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[1] Journal de l'Anatomie, 1879, p. 402.

[2] Centralb. f. Med. Wissensch., 1879, p. 145.

[3] Virch. Archiv, vol. 93, p. 318.

[4] Centralb. f. Med. Wissensch., 1882, p. 849.

[5] It is not probable that the *mean* lateral pressure in the arterial system can ever be raised above the normal degree in compensatory hypertrophy of the left ventricle; but it is quite conceivable and agreeable to clinical facts, that the *maximum* tension (attained at the close of the systole) may be excessive, and be counterbalanced, or more than counterbalanced, by undue diminution of tension during the ventricular diastole.

T. H., æt. 21, a warehouseman, came under treatment February, 1864, suffering from immense cardiac hypertrophy. The apex beat in the seventh interspace almost in the axillary line, and seven and a half inches from the mid-sternal base. The impulse was strong; the whole body shook at each beat of the heart. The pulses were visible in all the superficial arteries. A loud to-and-fro, roughish murmur was heard over the aortic cartilage, of which the diastolic part was greatly prolonged. This murmur was heard loudly at the base, but grew weaker toward the apex—beyond which it ceased to be audible. The valvular mischief seemed to be confined to the aortic orifice. There was no sign of serious mitral regurgitation nor any indication of impediment on the right side of the heart. There was total absence of a cyanotic tint; on the contrary, the face was pinkish pale, and the margin of the lips and tongue were of a faint rose; there was no swelling of the veins of the neck nor a trace of anasarca. On the other hand, the pulse was hard, resistant, bisferiens, ranging from 92 to 104. The character of the urine was highly significant. It was not high-colored and scanty, as in venous congestion, but abundant, pale, and of low specific gravity. The daily discharge varied ftom 57 to 65 ounces, the specific gravity from 1010 to 1015. It contained albumen, but only in small quantity; generally only a haze was produced with nitric acid; no tube-casts or other renal derivatives could be detected, though often looked for. It was distinctly observed that the proportion of albumen oscillated in accordance with the activity of the heart. When the ventricle was in high action the albumen rose; when it became more quiescent, under the influence of rest and digitalis, the albumen almost vanished for a time. I have recently seen this patient again (March, 1865) and find that his state is still as above described; there is a trace of albumen in the urine; but the general condition is wonderfully good.

The treatment of active renal congestion will be described with that of passive congestion, at the end of the next section.

PASSIVE CONGESTION.

The experiments of Robinson and Frerichs, already cited, show that an impediment or obstruction to the return of blood from the kidneys induces passive congestion of these organs, and, if sufficiently intense, causes albumen and blood to appear in the urine. An impediment of minor degree does not render the urine actually albuminous, but causes it to become scanty, high-colored, dense, and prone to deposit abundance of lithates.

Both these degrees of obstruction are frequently witnessed in clinical experience.

The obstruction may be seated in the chest, as in cases of valvular heart disease, emphysema, and pleuritic effusion: or in the abdomen, as when a gravid uterus or other tumor presses upon the emulgent veins or the upper course of the inferior cava. Sometimes a cirrhotic liver compresses the latter vein as it lies in the hepatic fossa.

The alterations on the side of the urine are not always proportional to the degree of obstruction to the circulation. Cases are met with, in which venous stagnation exists in an intense degree, with dropsy, orthopnœa, and pulsating jugulars, without a trace of albumen in the urine, and others in which the urine changes are strongly marked, while the more general symptoms of venous obstruction are only moderately so.

The two examples which follow, afford good illustrations of an unusual degree of renal derangement, secondary to obstruction to the circulation within the chest. In the first case the obstruction was due to old-standing tricuspid regurgitation; in the second to extensive emphysema.

Case 1.—A lawyer's clerk, æt. 44, came under observation December 6, 1862. He was suffering from œdema of the legs, ascites, and a severe bronchial attack. The features were livid; but the veins of the neck were not distended. The heart's apex beat in the fifth interspace, a little outside the nipple line. The cardiac dulness extended four inches vertically, and, about the same diagonally from base to apex. The heart's action was very irregular both in force and rhythm; pulse was 104. A loud blowing murmur was heard at the apex, of mitral regurgitant character, associated with a faint diastolic bruit, which was heard in maximum intensity over the second right costal cartilage. Loud bronchitic râles were heard universally over both lungs. There was copious mucous expectoration, sparsely speckled with blood; also severe dyspnœa, amounting at times to orthopnœa.

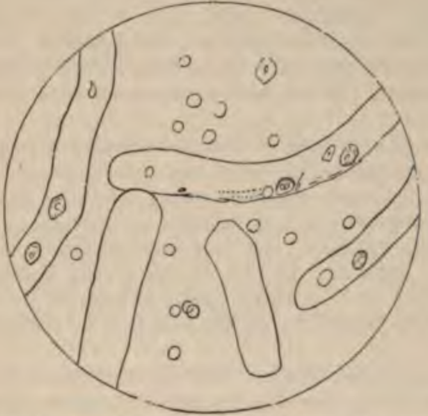
The urine was scanty, reddish, specific gravity 1025, with abundant clouds of lithates. It contained a small quantity of albumen (equal to about ⅛). The deposit, examined under the microscope (*see* Fig. 49), revealed numerous scattered blood-disks; casts of tubes, mostly perfectly hyaline, sometimes only visible when tinted with magenta; some casts were dotted with withered renal epithelia or with the nuclei of these; no oily or fatty particles were found.

On tracing back the patient's history, it appeared that he had had five attacks of acute articular rheumatism, of which the earliest occurred in his twentieth year. In one of these the heart had become affected. The cyanotic appearance and dropsical symptoms had shown themselves some months previously, but had suddenly assumed a formidable intensity a fortnight before, in consequence of a bronchitic attack.

With rest and other appropriate means, the bronchial attack subsided in about ten days. The dropsical and dyspnœal symptoms receded, and a moderately quiescent state was attained. The urine underwent corresponding change; it became more copious, its density fell, and the albumen faded to a mere trace; the casts remained as before.

While under observation this man went through three bronchitic attacks. In each, the urine went back to the character given of it in the first report. In the intervals again, the albumen became very scanty, and on two occasions the urine was found altogether free from albumen.

FIG. 49.



Casts of tubes and blood-corpuscles from the urine of a patient with passive renal congestion.

CASE 2—On October 16, 1862, a strongly built, stout woman, æt. 42, was admitted into the Manchester Infirmary, almost in a state of asphyxia. She was intensely blue in the face; could only breathe in the upright posture; her voice was a faint husky whisper; the tongue was livid; the veins of the neck were enormously dilated; she looked like a person half choked. There was not a particle of œdema nor any ascites; the limbs were firm and muscular. There was considerable drowsiness, but no actual coma. The sputum was frothy, not bloody. The examination of the chest revealed extensive capillary bronchitis, in emphysematous lungs; both bases were somewhat dull. There were no cardiac murmurs; the superficial cardiac dulness was inappreciable on account of the emphysema.

The urine was dusky red, and gave a play of colors with nitric acid (showing bile); no sugar in it; it was albuminous to a considerable degree ($\frac{1}{3}$). In the sediment, which was abundant and composed of lithates, were found numerous tube casts—nearly all hyaline; some of them studded here and there with altered epithelium, or altered blood-disks; a number of free cells were also found, most of them pus corpuscles, but some with solitary nuclei—evidently renal, and very little altered from their natural appearance. One cast was seen so studded with these as to deserve the name of an epithelial cast. Not a particle of fat was found in the renal derivatives.

On the next day but one (October 18) the patient seemed to breathe a little easier, but the surface was still intensely cyanotic. Of the urine, the notes state: "somewhat less highly colored; very much less albuminous, in fact the urine only becomes hazy with nitric acid; casts and cells still abundant."

Next day the breathing seemed again, if anything, rather easier, but the strength was evidently failing, and the drowsiness was becoming deeper. The urine no longer showed any bile tints, though still of a deep brown color. Albumen could only be discovered in it by very careful testing; the casts had all but disappeared; a few short fragments (slightly more granular than before) could with difficulty be found and identified. A few blood-corpuscles were seen after diligent searching.

October 20th.—There was evident emaciation going on, and steady diminution of strength. Scarcely any nourishment had been taken since admission; the voice was whispering, and the surface livid. The dyspnœal symptoms were at a standstill; drowsiness on the increase.

21st.—The urine was now quite free from albumen, and no casts could be found in the deposit. In the course of the succeeding night the patient quietly died, as if in sleep, partly exhausted by want of nourishment and the efforts to breathe, partly poisoned by the mephitic condition of the blood.

Autopsy, 18 hours after death. The *heart* weighed eleven ounces; the valves were healthy; a few slight atheromatous patches existed in the aorta. The *lungs* were in a state of excessive and universal emphysema; they bellied out of the cavities, when the chest was opened, like bladders of air. Spots of intense congestion were found here and there on section, and the extreme bases were somewhat œdematous. The *liver* was enlarged and congested. The *kidneys* were considerably enlarged, and weighed together twelve ounces; the capsules peeled off readily. On section the pyramidal and cortical substances were distinct from each other, and in due proportion; both parts were intensely congested, but otherwise natural, both to the naked eye and to microscopical examination. The body was still moderately well nourished; there was no anasarca in any part, nor any ascites.

There is one circumstance in this history which at first sight appears contradictory, namely, the disappearance of the albumen from the urine, notwithstanding that the obstruction in the chest persisted or even increased, and, indeed, brought the circulation ultimately to a standstill. The explanation of this occurrence is, I believe, to be found in the diminishing pressure in the arterial system from the gradual failure of the heart's power. Some of Robinson's experiments bear clearly on this point. He found, on ligaturing the renal veins in rabbits, that *vigorous* animals exhibited the urine changes (albuminuria, etc.) in far greater intensity than *weaker* animals; and he attributed the difference to the fact that in strong animals the powerful contractions of the ventricle served to maintain a greater counter-pressure on the arterial side of the renal circulation, and in this way intensified the intrarenal pressure or congestion. In the patient before us the pressure on the arterial side was visibly declining from day to day, in consequence of the inability to take food, which diminished the mass of the blood, and the progressive poisoning of the blood (from defective respiration) which gradually depressed, and finally annihilated the contractility of the ventricle.

The state of the kidneys in passive congestion varies with the duration of the obstruction. When the obstruction has been only recently established, as in the woman whose case has just been related, the kidneys are found simply enlarged and engorged; they resemble the kidneys of the rabbit, whose renal veins were ligatured by Robinson. They are dark in color, and on section a large quantity of dark blood exudes from the cut surface. Although the whole of the surface is redder than normal, yet the congestion is seen to be most marked in the pyramidal portion, where dark red, almost black streaks run from the apex to the base of each pyramid. The Malpighian corpuscles do not present the prominence seen in active congestion. On microscopic examination the veins and capillaries are found to be gorged with blood-corpuscles, while round the vessels red blood-corpuscles may be seen in the neighboring tissue, and even in the tubules themselves. The tubules in many places contain hyaline casts, and the epithelium is usually granular and sometimes fatty. But when the congestion has been in existence for months or years, the kidneys are found to have undergone far more profound alterations. The organs may then be somewhat smaller than normal, but are invariably hard and tough. The external capsule usually peels off easily, and leaves on removal a smooth or slightly granular surface. On section, the same congested state of the pyramidal portion is observed. The proportion of cortical to pyramidal substance is not much altered, but in very chronic cases the cortex may be somewhat atrophied. On microscopic examination, the most characteristic lesion is a great increase on the interstitial tissue between the tubules and round the glomeruli. This tissue is composed of fully formed fibrous tissue, containing but few cells; the basement membrane of the tubules is also thickened and passes into the thick interstitial tissue. The walls of the arteries are thickened, but there is no endarteritis seen. The convoluted tubules themselves are some contracted, some dilated, and their cells are usually small and cubical.

The exact nature of these latter alterations is a matter of dispute. Frerichs, Bergson, and Bamberger consider them as identical with those in the granular kidney of Bright's disease; but Traube contended that they were essentially different. Cornil is also of opinion that the lesion is not a nephritis.

Whatever anatomical difficulties there may be in the way of separating these cases from chronic Bright's disease; the clinical distinctions between them are clear and undoubted. Renal disorder from passive congestion comports itself quite differently from Bright's disease of independent origin, and from Bright's disease coming on in the course of chronic disease of bone or phthisis. The contrast between the first and the third is very marked. In the first (passive congestion), the renal affection has no momentum of its own, and makes no independent progress; it oscillates with the rising and falling intensity of the venous obstruction; it remains throughout a subsidiary complication of the primary disease, and assumes none of the special characteristics of Bright's disease (uræmia, etc.). On the other hand, when renal disease declares itself in the course of chronic phthisis, it assumes at once a formidable position. The entire clinical complexion of the case is transformed. Sometimes even the pulmonary disorder is altogether supplanted and thrown into the background by the more rapid progress and fatal course of the renal disease. (*See* case of M. C. in Chapter IV.)

TREATMENT OF CONGESTION OF THE KIDNEYS.—Congestion of the kidneys, whether active or passive, does not often call for separate treatment. Its course and intensity are usually contingent on the progress of the primary disorder of which it is a secondary phenomenon. But sometimes active congestion has an independent origin; in other cases, although secondary, it is sufficiently threatening to demand special attention. Passive congestion from cardiac and pulmonary obstructions can be most efficiently relieved by remedies applied to the primary complaints. But passive congestion from the pressure of a pregnant uterus—cases which will be considered at length in the appendix to this chapter—not unfrequently claims energetic treatment on its own account.

The most efficient means of combating active renal congestion are complete rest of the body, cupping the loins, brisk purgatives, the warm bath, and other diaphoretics. In the passive cases, cupping can only be of service when the congestion is due to a temporary cause, such as pregnancy; in the more common cases, the application of gentle counter-irritants to the loins is more serviceable, namely, tincture of iodine, embrocations, etc. Derivation by the bowels and skin is also an important means of relieving the overloaded organs.

APPENDIX.

On the connection of renal congestion, albuminuria, and Bright's disease, with pregnancy and puerperal eclampsia.

The late Sir J. Simpson was, I believe, the first to call attention to the occasional presence of albumen in the urine of pregnant women. The subject has since been studied on many hands with a view to elucidate the connection of the puerperal state, and especially of puerperal convulsions, with Bright's disease.

Albuminuria does not usually show itself in pregnancy until the seventh or eighth month, and often not until the approach of labor. Sometimes, however, it appears earlier, even so early as the third month. It is generally attended with œdema of the lower extremities, sometimes also of the face and upper parts of the body.

Blot found among the patients of a lying-in hospital, that one pregnant woman in five had albumen in the urine: this estimate is evidently very much too high for a general average. Abeille found, in private practice, the proportion to be one in ten; and Vans Arsdale and Elliott, one in fifty-six. ("New York Journ. of Med.," 1856.) This last estimate is probably the most nearly correct.

The albuminuria of pregnancy, and the accompanying anasarca, usually go on increasing up to the time of delivery, and then rapidly pass away. As a rule, the albumen is quite gone in forty-eight hours, sometimes even in twenty-four hours; but it may not wholly disappear for ten or fifteen days. If it continue beyond this last period, the gravest apprehensions of organic renal disease are justified.

The urine in the condition in question is usually scanty, of high specific gravity and dark color, contains a large quantity of albumen and a little blood, and deposits hyaline tube-casts and urates on standing. Hiller found in one case crystals of hæmatoidin.

If we inquire into the origin of albuminuria in pregnancy, two conditions present themselves, which, altogether or separately, are capable of explaining its occurrence: these are (*a*) alterations in the circulation of blood through the kidneys, and (*b*) the alterations in the quality of the blood which are proper to the pregnant state.

The older observers, and perhaps the majority of the modern writers, are of opinion that the principal change in the circulation of the kidney is passive congestion due to pressure of the uterus on the renal veins. According to this view, the growing womb mounting into the abdomen necessarily exercises a certain compression on the contents of that cavity, and among other structures, on the inferior cava and renal veins. This mechanical pressure occasions a passive congestion of the kidneys, which, if sufficiently severe, induces albumen to appear in the urine, together with blood and tube-casts. That this is one of the most efficient causes of albuminuria in pregnancy, is indicated by the fact that primiparæ, in whom the parts are resistent, and the pressure therefore intense, are disproportionately liable to albuminuria; so, too, albuminuria is common where the liquor amnii is in excess; and in twin pregnancies also albumen does not usually appear in the urine until the later periods of gestation, when the venous stagnation has reached its height.

The recent observations of Leyden have, however, caused doubts as to the correctness of the above view. From the results of several autopsies he showed that the kidney if examined soon after delivery was large, the cortex swollen, pale and *anæmic*, and that the epithelium had undergone advanced fatty change. When examined at a greater interval from the delivery, the kidney was still pale, but had lost its swollen and fatty condition.

The urine, too, differs from that of venous congestion of the kidneys, in containing a large instead of a small quantity of albumen.

Leyden's description of the kidneys has since been confirmed by Hiller, but the cause of the anæmia of these organs is not clear.[1]

Flaischlen has suggested that the anæmia may be due to irritation from the uterus acting reflexly on the vasomotor system and causing constriction of the renal vessels. Numerous other views have been put forth, but at present a groundwork of facts is wanting.

The altered condition of the blood probably contributes more to the establishment of the œdematous swellings, especially in the upper parts of the body, than the mechanical pressure. The blood, in pregnancy, is poor in red corpuscles and more watery than natural—a condition highly favorable to serous transudation, and to the production of anasarca and albuminous urine.

The expulsion of the fœtus is, as has been stated, commonly the signal for the disappearance of the œdematous swellings, and the restoration of the urine to its healthy state; but in a certain number of cases eclamptic convulsions break out about the time of parturition, and of these about 30 per cent. prove fatal; in certain other cases the albuminuria does not disappear after delivery, but persists, with or without dropsical effusions, until there is no longer any doubt that genuine and confirmed Bright's disease has been established.

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[1] Dr. Dickinson, in his work on Albuminuria, describes cases of the albuminuria of pregnancy in which the kidneys were found pale and fatty, but he also gives a plate representing the kidney in another case, where venous congestion was well marked.

There has been much dispute as to the exact nature of the connection between the events here enumerated and the puerperal state. It has been on the one hand alleged, and on the other hand denied, that the pregnant state is an effective exciting cause of Bright's disease; it has also been both alleged and denied that puerperal convulsions are of renal (uræmic) origin.

There can be no doubt that many of the cases in which Bright's disease coexists with, or follows, the pregnant state, are examples of the coincidence of two mutually independent conditions. Pregnant women are, of course, liable, like other persons, to contract Bright's disease from any of its ordinary causes; and, again, women who are already the subjects of Bright's disease may become pregnant. But after eliminating the cases belonging to these two categories, there are still, as I believe, a considerable number in which Bright's disease has been really *caused* by pregnancy. The Registrar-General's reports furnish some valuable evidence on this point. In the five years 1857–61, there were registered 6220 deaths from Bright's disease. Of these 3699 were males, and 2521 females—being in the proportion of 68 females to every 100 males; this was the relative proportion between the two sexes at all ages. But the deaths of women from Bright's disease during the child-bearing years of life (from twenty to forty-five) far exceeded this proportion—being as high as 80 women to every 100 men. After the age of 45, the proportion of deaths from Bright's disease sank again to 59 women for every 100 men. There seems no other conclusion to be drawn from these numbers, than that the puerperal state is a prolific cause of Bright's disease.

A certain number of pregnant women having albuminuria (probably about one in ten) are affected with epileptiform convulsions (or eclampsia) before, during, or after labor.[1]

It is a question of considerable interest whether puerperal eclampsia is due, or not, to uræmic poisoning. The affirmative has been warmly supported by Frerichs, Braun, Litzmann,

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[1] The liability of albuminuric pregnant women to eclampsia is estimated at a much higher rate than this by Blot, Meyer, and Devilliers and Regnauld. The united statistics of these observers give a proportion of about 1 in 4. Taking all pregnancies together—with and without albuminuria—about 1 in 500 are complicated with eclampsia. Braun gives the proportion as 1 in 545.

Wieger, and others; and the negative by Scanzoni, Depaul, Rosenstein, and several more.

On the affirmative side it has been shown: (1) that eclamptic fits are similar, symptomatically, to uræmic convulsions; (2) that albumen is almost invariably present in the urine of eclamptic patients; and that in several cases undoubted evidence of organic kidney disease has been found after death; (3) that, frequently, anasarca of the upper parts of the body, and dryness of the skin, coexist with albuminuria, and confirm the diagnosis of Bright's disease.

On the opposite side it is alleged: (1) that there are authentic instances (apart from epilepsy, apoplexy, or hysteria) of puerperal eclampsia without albuminuria;[1] (2) that anatomical evidence of Bright's disease has only been found in a minority of the cases; that more frequently the kidneys have only been found congested without any organic alterations which could be identified with any form of Bright's disease; (3) that other causes (than Bright's disease) have been repeatedly found in the bodies of persons dying of puerperal eclampsia, namely, œdema of the brain, and congestion of the meninges, which probably were not without concern in bringing about the attacks.

The want of segregation of irrelevant cases prevents the possibility of a clear analysis of the facts adduced in this dispute. But it is evident that the existence of well-attested cases of eclampsia without a trace of albumen in the urine, is fatal to the *universality* of the uræmic theory. On the other hand, the not infrequent coexistence of undoubted Bright's disease, leads strongly to the conviction, that, in many cases, the convulsions are truly uræmic. It must not, however, be forgotten, that pregnant women who are the subjects of confirmed Bright's disease frequently pass through labor without the least convulsive disturbance.

As the evidence now stands, puerperal eclampsia cannot always be attributed to one and the same invariable cause. In some instances, the convulsions appear to be essentially of a reflex character, arising from irritation of the generative organs, acting on a nervous system in a state of exalted sensibility. It is at the period when this sensibility attains its maximum, namely, during the act of labor, that convulsions usually break forth. But it is likewise about the same period that the pressure within the abdomen becomes most intense, and the stagnation in the renal veins and interruption to the secretion of urine most complete. When the act of birth commences there are added to these causes of disturbance, violent and general muscular contractions causing suffusion of the features and congestion of the cephalic meninges. Several explosive elements are thus brought together at the same period; and it is scarcely to be wondered at, that the equilibrium of the nervous system should be thereby occasionally overset.

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[1] Among other examples, the following may be referred to: Abeille, loc. cit., p. 607; Riedel, Zeitschr. f. d. Geburtscheilk., 1858, p. 13; Rossi, ibid., 1863, Bd. ii. S. 72. *See*, also, Schröder, Ingerslev, and Löhlein, quoted by Wagner, loc. cit.

The recognition of two or more categories of puerperal eclampsia, is of much importance both for prognosis and treatment; and the want of some rational classification of the cases is doubtless one cause of the discrepancies in the experience of different observers as to the beneficial effects of venesection and other plans of treatment.

At least three categories seem to deserve to be recognized, viz., 1. *Cases depending on confirmed and chronic Bright's disease.* In these the eclampsia must be regarded as mainly or wholly uræmic; the ultimate prognosis is lethal, and depletive measures are less indicated than chloroform, etc. 2. *Cases depending on passive congestion of the kidneys, or on a condition resembling, if not identical with, acute Bright's disease.* These are usually primiparæ; the phenomena are probably partly uræmic, and partly reflex; the prognosis is favorable, were the fits once over; active depletive measures are indicated. 3. *Cases depending on reflected uterine irritation and meningeal congestion.* In these the urine is *not* albuminous; the prognosis is favorable, were the fits over; they call for active depletory measures.

Something remains to be said in the way of diagnostic indication, in cases of pregnancy complicated with albuminuria.

The urine of a pregnant woman being found albuminous—how shall it be known, whether there exists confirmed Bright's disease or only a temporary disorder which will harmlessly subside after parturition? The following points tell strongly for confirmed Bright's disease—an abundant flow of pale urine of low density; presence of granular or fatty casts, a considerable amount of albumen and yet a relaxed state of the abdomen and tissues generally; anæmia; a markedly hypertrophied left ventricle; anasarca equally distributed (or nearly so) over the whole body. The points which, on the other hand, tell in favor of congestion, or of acute (and curable) Bright's disease are: evident signs of severe pressure within the abdomen; the patient being a primipara; the quantity of albumen in the urine appearing to bear a proportion to the existing venous congestion; the urine being high-colored, scanty, and dense; the anasarca being mostly, or altogether, confined to the lower extremities; absence of anæmia and cardiac hypertrophy.

By attention to these points I have been able, in the cases which have fallen under my observation, to frame a diagnosis which the event has justified.

CHAPTER II.

BRIGHT'S DISEASE.

PRELIMINARY REMARKS.

Cases characterized by albuminuria and dropsy, depending on structural changes in the kidneys, are classed together under the general title of Bright's disease.

Several different pathological states are doubtless included under this designation; and the cases present considerable diversity, not only in the acuteness of their course but also in their modes of origin and symptoms. Numerous attempts have been made to divide and classify the various conditions of the kidney found after death from Bright's disease; and to connect each with its appropriate clinical history. Hitherto none of these attempts has obtained general assent; and a regrettable confusion of nomenclature has been added to the inherent intricacies of the subject. Notwithstanding the diversities just referred to, the points of resemblance between the several varieties of Bright's disease are so strong and so numerous, that they form an easily recognized clinical group. This resemblance arises, in great part, from the circumstance that the structural changes in the kidneys, various as they may be, bring about the same ultimate results, namely, impoverishment of the blood from loss of albumen, with poisoning of it from retention within the body of the excrementitious matters of the urine; and the more prominent symptoms in Bright's disease arise from this changed condition of the blood, rather than from the *direct* effects of the structural changes in the kidneys.

Opinions are divided, in the first place, as to whether there be a fundamental unity beneath the apparent diversity; in other words, whether the "large, smooth, white kidney," the "small, smooth kidney," the "granular uncontracted kidney," and the "granular contracted kidney," are successive stages of one and the same pathological process, or represent radically distinct diseases.

Dr. Bright, whose researches on this subject have made his name so renowned in medical science, expresses himself quite doubtfully on this point. In his introductory remarks to the twenty-three cases first published by him, in 1827, he says:

"From the observations which I have made, I have been led to believe that there may be several forms of disease to which the kidney becomes liable in the progress of dropsical affections. I have even thought that the organic derangements which have already presented themselves to my notice, will authorize the establishment of *three* varieties, if not of three completely separate forms of diseased structure." But toward the close of the same remarks he observes: "Although I hazard a conjecture as to the existence of these three different forms of disease, I am by no means confident of the correctness of this view. On the contrary, it may be that the first form of degeneracy to which I refer never goes much beyond the first stage; and that all the other cases, together with the second series and the third, are to be considered only as modifications, and more or less advanced states of one and the same disease." (Reports, pp. 67 and 69.)

Soon after the period when these sentences were written, a new vantage ground for the study of renal diseases was acquired by the researches of Mr. Bowman, which threw a strong light on the intricate anatomy of the kidney. Histologists of eminence both in this country and Germany—Busk, Toynbee, Simon, Henle, Rokitansky, Virchow—and inquirers who have made the subject a special study—Johnson, Frerichs, Basham, Dickinson, Grainger Stewart, and many more—have worked with unexampled perseverance to ascertain the nature and arrange the varieties of the morbid processes taking place in the kidneys in Bright's disease; and yet the doubt which possessed the mind of Bright has not been wholly cleared away.

All this labor has not, of course, been thrown away. On the contrary, much light has been shed on the pathology of the complaint; and data of importance have been obtained for prognosis and treatment. More especially the examination of the organic admixtures of the urine—renal epithelium and casts of the uriniferous tubes—has yielded to Dr. Geo. Johnson results of the highest clinical value, which claim for him a pre-eminent mention in this field of pathology.

Frerichs considers that Bright's disease is essentially one, and that it is of an inflammatory nature. He divides the anatomical changes in the kidneys into three forms, which he regards as stages of the same fundamental process, namely:

1. The stage of hyperæmia and commencing exudation.
2. The stage of exudation and commencing change of the exudation.
3. The stage of degeneration and atrophy.

Dr. Johnson, on the other hand, recognizes several distinct processes under the common heading of Bright's disease—but chiefly two, and both of an inflammatory nature—one characterized by a shedding and destruction of the epithelial lining of the uriniferous tubes (*desquamative nephritis*), and one without such desquamation, and affecting the intertubular structures of the organ (*non-desquamative nephritis*). He also gives a separate place to "fatty degeneration" and "waxy degeneration" of the kidney.

Dr. Dickinson divides Bright's disease into three main varieties. 1. *Tubal nephritis*, in which the uriniferous tubes are the seat of an inflammatory action. This he subdivides into an acute and a chronic form. 2. *Granular degeneration*, in which there is increase and subsequent contraction of the intertubular matrix of the kidney. 3. *Depurative disease*, which is the name he gives to amyloid or waxy infiltration of the kidneys.

Dr. Grainger Stewart suggests the name "Bright's Diseases" as more truly descriptive than the old designation "Bright's Disease." He classifies the cases as follows:

1. *The inflammatory form*, of which there are three stages—
   a. That of inflammation.
   b. That of fatty transformation.
   c. That of atrophy.
2. *The waxy or amyloid form*, of which also there are three stages—
   a. That of degeneration of the vessels.
   b. That of secondary changes in the tubes.
   c. That of atrophy.
3. *The cirrhotic, contracting, or gouty form.*

In addition to these he described two mixed types, in which in the one case the waxy form, and in the other the cirrhotic form, is combined with the inflammatory form.

It would lead me too far to discuss the merits of these and the many other classifications which have been put forth. I content myself with simply indicating the more important ones. In the following pages the subject will be treated from a clinical, rather than an anatomical, point of view, and the cases will be classified under the two main heads of *acute* and *chronic* Bright's disease. The former embraces a compact and universally recognized group, which formerly went under the designation of "inflammatory dropsy." It corresponds to the acute desquamative nephritis of Johnson, to the first stage of Frerichs, and to the acute tubular disease of Dickinson. The latter includes the protracted cases, which either have lapsed into a chronic state from the acute form, or, which is far more frequent, have been chronic from the beginning. Three types of chronic Bright's disease will be recognized. 1. Cases which have lapsed from the acute state (kidney smooth, white, generally large, exceptionally dwindled). 2. Cases which have been chronic from the beginning (kidney granular, red, contracting). 3. Cases associated with waxy or lardaceous (so-called amyloid) degeneration of the kidneys.

The presence of fat in the renal substance, and in the epithelium of the tubes, is not special to any one type of renal degeneration; but is found associated with anatomical changes of the most varied kinds: it has therefore no claim to a separate consideration.

GENERAL ETIOLOGY OF BRIGHT'S DISEASE.

The special etiology of the several types of Bright's disease will be separately considered in the twe following chapters, but it will be convenient in this place to consider some of the points bearing on the etiology of Bright's disease as a whole.

The want of uniformity in our nomenclature of organic diseases of the kidneys has considerably lessened the value of the returns of the Registrar-General in this field of pathology. Cases registered on the certificate of death as "Bright's disease" are entered in these returns as "nephria;" but it is evident that the larger number, even of the cases recognized as Bright's disease during life, are not so registered, but are classified under the heads "nephritis" and "kidney disease." To obtain some idea of the prevalence of Bright's disease, let us take the numbers under these three designations. There were registered in England and Wales, in 1868:

2076 deaths from "nephria,"
495 deaths from "nephritis,"
2836 deaths from "kidney disease,"

—making a total of 5407. This yields only a proportion of 1.1 per cent. of the total deaths from all causes—a number which is probably considerably below the true proportionate mortality from Bright's disease. Without admitting, with Mr. Simon, that two-thirds of the cases of Bright's disease run a latent or undiscovered course, it must be allowed that a very large number are overlooked in these returns, and are probably to be found among the 6284 entered as "dropsy," or among those entered under "convulsions," "pneumonia," and other headings.[1]

Bright's disease is about one-third more common among men than women (1215 men to 861 females). The excess of deaths among males, although present at every age, is not equal at the different periods of life: it is most marked between the ages of forty-five and sixty-five.

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[1] It is evident, however, that Bright's disease is gradually becoming better known in this country, and more frequently identified. In 1852, only 570 deaths were entered under "Nephria." In each successive year the number rose, quite out of all proportion to the increase of the population, until in 1861 it reached 1148, nearly thrice as many as in 1852; and in 1868 it reached 2076, nearly four times as many as in 1852. Correspondingly, the entries under "dropsy" diminished, from 9788 to 7301, and to 6284 for the same three years.

The mortality from Bright's disease shows a progressive increase from childhood up to about the age of 50; in the succeeding 20 years the mortality continues steady, at a somewhat lower, but still high, rate; the next decade shows a decided diminution as regards Bright's disease, though the general mortality at this epoch is at its highest point. These facts are exhibited in the following table:

TABLE *showing the number of deaths registered as "Nephria" (Bright's disease) in England and Wales in 1868, at the different periods of life:*

	Under 5 yrs.	5-15 yrs.	15-25 yrs.	25-35 yrs.	35-45 yrs.	45-55 yrs.	55-65 yrs.	65-75 yrs.	75 years and upwards.	Total at all ages.
Males . .	41	60	87	157	216	247	225	133	49	1215
Females .	34	39	68	133	160	147	148	97	35	861
Both sexes .	75	99	155	290	376	394	373	230	84	2076

That complex of impressions which is familiarly known as taking *cold* is the common cause of Bright's disease in its acute form. *Cold*, operating more slowly and continuously, also constitutes a prolific source of chronic Bright's disease. Persons whose occupation exposes them to cold, wet, and the inclemencies of the seasons, without adequate protection—those who work in hot workshops, are in the habit of going to cool their reeking bodies in the open air—the indigent classes, who dwell in damp cellars, insufficiently clad and ill-fed, amid dirt and squalor, furnish a large quota of victims to this disease.

Dr. Johnson is at especial pains to explain the *modus operandi* of this frequent cause of renal disease. He contends that the defective action of the skin causes certain deleterious matters to accumulate in the blood, and that the burden of their elimination is thrown upon the kidneys, which receive injury thereby. It is impossible to accept this view without great limitation, seeing that suppressed cutaneous transpiration ushers in a multitude of inflammatory and febrile conditions, without provoking renal disease. When a person "takes cold," it is a fact that the secretion of the skin is very much diminished or altogether suppressed: but it is not possible to predicate on what organ the injurious impression will ultimately settle—whether on the bronchial tubes, the pleura, the lung tissue, the kidneys, or some other organ or part of the body; so that it cannot be maintained that there is any *special* relation between suppressed cutaneous secretion and the genesis of renal disease.

The abuse of spirituous liquors ranks high as a determining cause of Bright's disease. Christison estimates the proportion due to this cause, in Edinburgh, as three-fourths or four-fifths of all the cases; and he justly remarks that it is not habitual drunkards only who show this tendency to renal disease, but dram-drinkers, who are in the constant practice of using ardent spirits several times in the course of the day, without becoming actually intoxicated.[1]

Malt liquors—though far less pernicious than spirits—are not without influence to produce Bright's disease when largely indulged in. In a journeyman baker, under my care at the Infirmary, the disease was clearly traced to the habit of fuddling himself with beer from Saturday evening to Monday morning, which the patient had followed for several years.

Very frequently, intemperate habits go hand in hand with a grimy skin and exposed occupation; and the subjects of Bright's disease are found disproportionately numerous among laborers, well-sinkers, cabmen, carters, hawkers, glass-blowers, smelters, and puddlers.

A large number of cases arise in connection with some *constitutional vice*, more especially tuberculosis or struma, and chronic lead poisoning. Among the easier classes, gout and constitutional syphilis are prominent antecedents.

It seems now to be fully attested that *ague* may cause Bright's disease in its various forms.[2]

Chronic affections of the lower urinary passages (cystitis, stricture, etc.) frequently lay the foundations of renal disease. In a boy of seven, who died in the Royal Infirmary, a small stone no larger than an almond was found lodged near the neck of the bladder. Repeated sounding had failed to detect it during life; operation was consequently not performed. For some weeks before death, general anasarca had shown itself. The kidneys were found wasted to an extreme degree; the cortical substance was reduced to a thin edge no thicker than a shilling; the pelvis of the kidney and the ureters were dilated, and their lining membrane thickened and bathed in pus. In cases of this class there is a double influence tending to produce renal degeneration, namely, the long-continued exhausting suppuration and direct transmission of the inflammatory process by continuity of tissue.

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[1] Dr. Dickinson, in his work on Albuminuria, has, in an elaborate chapter, called in question the efficiency of intemperance as a frequent cause of chronic Bright's disease. I have examined his statements at length in a paper in the Brit. Med. Journ. for Nov. 4, 1871,—and have shown, conclusively as I believe, that the arguments he advances are inadequate to shake the old opinion.

[2] A full account of the anatomical changes produced in the kidney by ague will be found in a paper by Kiener and Kelsch, Archives des Physiologie, Feb. 1882.

The use of mercury, which Wells and Blackall believed capable of producing albuminuria and renal mischief, has not been found by observers of wider experience to have this effect. Rayer and Desir, out of forty cases treated with mercury at the Hôpital des Vénériens, only found a slight quantity of albumen in two—in both of which its presence was accounted for by the existence of pus in the urine. Rayer further observes that he had for years used a multitude of mercurial preparations in the treatment of various diseases without ever having observed the production of dropsy. He also states that he had treated a large number of gilders affected with mercurial trembling, and that he had not seen a single case of dropsy with coagulable urine supervene during or after this trembling (*see*, also, p. 196, note).

A certain number of cases of chronic Bright's disease present themselves, in which the most searching analysis fails to indicate the exciting cause of the disorder. In some of these the renal affection is only a part manifestation of some widespread cachexy, as an example (M. H.) to be related in Chap. IV., in which fatty degeneration coexisted in the heart, great vessels, brain, and kidneys.

CHAPTER III.

ACUTE BRIGHT'S DISEASE.

Synonyms—Inflammatory Dropsy; Diffuse Nephritis; Acute Desquamative Nephritis (Johnson); *Acute Tubal Nephritis* (Dickinson).

ANATOMICAL CHARACTERS.—The kidneys are always more or less enlarged—sometimes to twice their natural size; their surface is smooth; the capsule thin, transparent, and easily stripped off; their color varies; it is generally a deep dusky red; but sometimes a light fawn, almost white; in other cases it is mottled red and white. The superficial veins are larger and more distinct than natural. When the kidney is cut open the cortical substance is found to be increased very much out of proportion to the pyramidal. The *red* congested kidney exudes a bloody sanies abundantly from the cut surface; a number of hemorrhagic spots may be generally seen scattered through the cortex or beneath the capsule. The surface of the section is dusky red, and studded with minute darker-red points, which are the engorged Malpighian corpuscles. The *pale* and the *mottled* kidneys present a contrast of color between the cortex and the pyramids. The latter appear unnaturally red, and from their bases radiating lines of red spread, fan-like, into the cortical substance. The cortical portion is smooth and white, or yellowish-white, and spotted like ivory.

Under the microscope almost all parts of the kidney may be found affected, but in varying proportions in different cases. Usually the chief alteration is situated in the epithelial contents of the convoluted tubes. The diameters of these are increased, and in extreme examples to twice or even thrice their normal measurement. The epithelial cells are also increased in size, they cannot easily be distinguished one from another, and their free extremities are rounded. The striation of the protoplasm at the base of each cell is no longer seen, but instead an extremely granular condition is observed, which in advanced cases gives place to a collection of fat globules. The nuclei of the cells may be hidden by the swollen protoplasm; when seen, they are found to be greatly proliferated, and a division of the cell substance taking place round each nucleus, we have a multiplication of the cells.[1] Cornil has described in the cell-protoplasm certain hyaline globules, which apparently proceed from the base to the free margin and discharge themselves into the lumen of the tubule. The epithelial cells may be so hypertrophied as to fill completely the lumen of the tubule. More commonly the lumen is occupied either by accumulations of cells, consisting of detached epithelium and leucocytes, or by a fibrinous exudation, which in the higher tubules assumes a net or star-like arrangement; but lower down, and especially in the straight tubes, it forms glossy cylinders of various size, according as the tubes have preserved or have shed their proper lining. In the straight tubes the epithelium gives evidence of similar changes, but to a much less intense degree. Their larger bore and direct course favor the escape of the detached epithelium, so that some of them are partially or wholly denuded. In the later stages the cells of the convoluted tubules also break down, and Klein has described, in scarlatinal kidneys, a deposit of lime salts in the cells.[2]

The interstitial tissue of the kidney may be unaffected, but often shows an infiltration of leucocytes, even at a comparatively early period. These are situated most commonly round the glomeruli constituting the so-called "pericapsular" nephritis; but later on cell-accumulation is found also between the tubules and around the arteries.

The arteries and capillaries are distended with blood-corpuscles, and more or less extensive rupture of the capillaries takes place. The hemorrhages sometimes form a marked feature. Masses of red blood-corpuscles are then found lying in the intertubular tissue, distending the tubules themselves, and occasionally lying between the epithelial cells or embedded in the cell-protoplasm. Klein has described a hyaline thickening of the intima in the arteries, and more especially in the afferent vessel of the glomerulus. The nuclei of the endothelium and of the muscular fibres he also found in a state of proliferation.

The morbid process seems to consist essentially in a catarrhal condition of the uriniferous tubes, with a prodigious swelling and proliferation of their epithelial elements. At the first, there is an inflammatory congestion of the organs with rapid swelling and rupture of the capillaries. On that follows increased production of epithelial cells; these multiply, choke up and distend the uriniferous tubes, thereby compressing the renal capillaries and impeding the circulation through them.

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[1] A few observers, and most recently Cornil and Brault, have denied that such proliferation of the epithelium takes place, when the inflammation attacks a previously healthy kidney.

[2] Litten also has described calcification of the cells. *See* Virch. Archiv, Bd. 83, S. 508.

[3] Many of the appearances described above have been made use of by some authors as the bases of a classification. Thus, Wagner describes four forms of acute Bright's disease. 1. The hemorrhagic-catarrhal. 2. The hemorrhagic-catarrhal with interstitial change. 3. The acute large pale kidney. 4. The acute lymphomatous kidney. It has not been thought advisable in a work like the present to attempt any classification of the anatomical features.

When this proliferation has reached a certain degree, the kidneys, which before were of a dusky red, become pale or mottled—not so much from an actual deficiency of blood in the organs, but rather, as Dickinson explains, from the white color of the masses of epithelium overpowering the natural red of the parts.

The choking-up of the tubes with their own epithelium necessarily impedes the depurating functions of the kidneys, and the blood is poisoned with excrementitious matters. The urine becomes scanty in amount, and deficient in its proper constituents; it carries with it, as it percolates the diseased ducts, loose epithelium, blood, and fibrinous exudation, or detaches whole tracts of the lining, all of which objects form an abundant grumous sediment in the urine.

How soon the change from red to white takes place, depends on the rapidity of the multiplication of the epithelial cells. I have seen the bloodless condition reach an extreme degree in six weeks. Dr. Dickinson states that it may occur within four days.

During recent years the attention of pathologists, and also of clinical observers, has been turned to the changes undergone by the glomeruli in certain cases of acute and subacute Bright's disease. When the conditions described above are present, the glomeruli are seen on the cut surface of the kidney, as red points; while under the microscope, the capillaries of the Malpighian tuft are found injected, and the space between the tuft and the capsule filled with red blood-corpuscles. In the cases of so-called glomerulo-nephritis the glomeruli appear as pale points on the section; and extensive inflammatory changes are found in them, causing almost total abolition of their secreting power, while in the remainder of the kidney little or no change may be seen. The results of different observers show considerable variance with regard to the exact changes found in the Malpighian corpuscle, but they may briefly be stated as follows:

Most frequently an accumulation of cells is seen between the capsule of Bowman and the glomerular tuft. These cells consist of a few leucocytes escaped from the vessels, but for the main part, of cells resulting from a proliferation of the epithelial lining of Bowman's capsule. Proliferation has also been observed in the thin layer of epithelium covering the tuft and dipping between its divisions, and doubtless this contributes to the cell accumulation in the glomerular cavity. My colleague, Dr. Leech,[1] has described proliferation of both these layers in a case under his observation, and he was able also to trace the development of the cells into a mass of fully formed fibrous tissue filling up the glomerular space. The glomerular tuft is compressed by the new growth, and the circulation through it is stopped. Hence, its pale appearance when a section of the kidney is made. But apart from any exudation between the tuft and the capsule, the circulation through the glomerulus may be interfered with by changes in the tuft itself. These changes are generally evident from the great increase in the number of nuclei in the tuft. The numerous nuclei may in part be due to a proliferation of those portions of the epithelial covering of the tuft which dip down between its several divisions. They may also be the nuclei of leucocytes escaped from the vessels. Klebs believed that they were due to inflammatory proliferation of the connective-tissue cells binding together the divisions of the tuft. This view has received but little support, and, indeed, the existence of connective tissue in the normal tuft is denied. Another source of the numerous nuclei has been found in a proliferation of the nuclei of the capillaries themselves. Klein has shown that the capillary walls may be swollen and show the same hyaline change he has described in the afferent artery of the glomerulus. Friedländer, too, has described changes in the interior of the capillaries of the tuft. In his cases the circulation through the tuft was stopped, not by pressure from without, for the vessels of the tuft were even larger than normal; but by blocking of the capillaries with a granular mass containing nuclei, and produced, as he thinks, by a thickening of the capillary wall.[2]

In the acute nephritis which accompanies or follows the infectious diseases, groups of microörganisms have been seen in the kidney. They may be situated in the bloodvessels, causing a thrombosis; or they may be seen in the urinary tubules, and within the epithelial cells.

COURSE AND SYMPTOMS.—The invasion of the disease is commonly abrupt, and traceable to some definite cause. A person takes cold, or falls into a fit of intemperance, and next morning, or in two or three days, the face begins to swell, then the hands and body generally. In another large class of cases the disease breaks out during convalescence from scarlet fever or—much less frequently—some other febrile or zymotic complaint.

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[1] On Glomerulo-nephritis. Brit. Med. Journal, 1881, i. p. 994.

[2] The principal works on Glomerulo-nephritis are the following:

Klein, Reports to the Privy Council, 1876, p. 39. Greenfield (Atlas of Pathology, Syden. Soc.). Waller, Journal of Anat. and Phys., vol. xiv. p. 432. Leech, Brit. Med. Journ., 1881, vol. i. p. 994.

Klebs, Handb. d. Path. Anat., vol. i. p. 645. Langhans, Virch. Arch., vol. lxxvi. p. 85. Litten, Charité Annalen, vol. iv. p. 30. Friedländer, Fortschr. d. Medicin, vol. i. No. 3.

Acute Bright's disease is usually ushered in with chilliness or shivering, headache, nausea, vomiting, pains in the back and limbs, arrest of the cutaneous perspiration and oppression in the chest.

Dr. Mahomed has made important observations on a pre-albuminuric stage of Bright's disease following scarlatina. He states that in the desquamative stage of scarlet fever, certain symptoms arise which are premonitory of the appearance of albumen in the urine, namely, high tension of the arterial system as measured by the sphygmograph, and transudation into the urine of the blood-crystalloids as tested by guaiacum and ozonic ether. The almost universal forerunner and probable cause of these symptoms, he says, is constipation. One day passed without an action of the bowels is sufficient to give rise to them. If a sharp purgative be administered these symptoms pass away; if not, they are succeeded by the usual signs of severe renal disturbance.

When fairly established, the symptoms are exceedingly distinctive. The countenance is pale and puffy, with a heavy stupid expression; the limbs and trunk are anasarcous. The œdematous parts are resistant on pressure, and pit little or none. More or less effusion takes place into the serous cavities, especially the pleura and peritoneum.

There is a general febrile movement; the pulse is hard, full, and of high tension, the appetite lost, thirst excessive; the skin is dry, and the whole surface blanched and tumefied. An uneasiness or dull pain is felt in the loins, and the renal regions are tender on pressure.

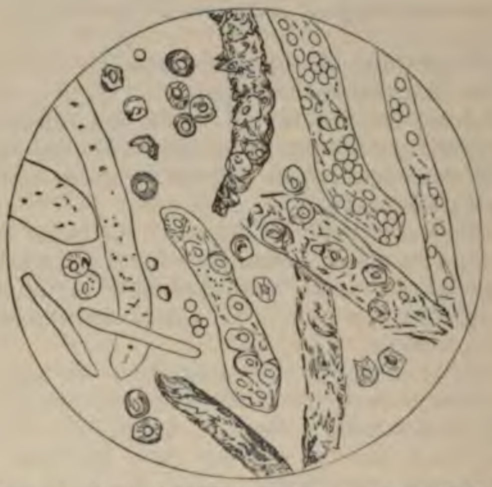
The *urine* is of a smoky or dusky hue—in some instances dark brown like porter—from the presence of altered blood. On standing, it deposits a copious, flocculent, dirty-brown or chocolate sediment, like the settling from beef-tea. It is very albuminous; it may even become quite solid on boiling. The specific gravity, in the stage of increment, is usually above 1020, often much higher, mounting sometimes to 1030, and in one instance which occurred to me even to 1065. When of high density, the urine is proportionally scanty; it may not exceed 12 or 18 ounces in the twenty-four hours; in extreme cases it may sink to 4 or 6 ounces, or be, for two or three days, altogether suppressed. The calls to void it are more frequent than in health, especially at night and in the recumbent posture; the patient has to get up three or four times in the course of the night to empty the bladder. The urine is generally acid, and surcharged with pigment; it often deposits the amorphous urates. Very rarely it is alkaline from fixed alkali. The natural urinous odor is lost; it has a faint unpleasant smell, which has been compared to that of the washings of flesh.

The deposit when examined microscopically (see Fig. 50) is found to consist of blood-corpuscles, loose renal epithelium, free nuclei of these, tube-casts, shapeless masses of coagulated fibrine, and the broken débris of all these structures.

There are also generally found epithelial cells from the pelvis of the kidney and the bladder.

The renal epithelia vary a good deal in their appearance. Sometimes they look almost natural, only somewhat swollen and opaque. More frequently they are much broken down; their nuclei are set free, or are only invested in part by the granular cell-contents which naturally surround them. The disintegrated epithelium forms an amorphous dark granular débris scattered over the field. When very abundant, the epithelium communicates a milky appearance to the urine. The free nuclei greatly resemble red blood-disks both in shape and size, but they are devoid of the biconcave figure, and refract light more strongly. A solution of magenta tints them of a deep carbuncle-red. The free blood disks are frequently distorted. When the urine is of high density, they are shrunken, and often puckered at the margins; on the other hand, when the urine is of a lower density, 1017 and under, they expand, lose their central depressions, and eventually burst, and cease to be recognizable.

Fig. 50.



Transparent, granular, blood and epithelial casts from a case of acute Bright's disease; free renal epithelium; and blood disks.

The tube-casts are abundant, and of varied size and appearance. The most common are of "medium" size, transparent, beset with epithelial cells or blood disks. Mixed with these may be some "very large" and some "very small" hyaline casts, together with opaque granular casts (Fig. 50).

Specks of oil are generally altogether absent; sometimes, however, a few small ones are seen either on the casts or within the epithelia; but their number is always quite insignificant in the early stages of the disease.

The proportion of albumen in the urine during the height of the complaint varies, according to Frerichs, from 8.2 to 12.7, 17.5 and 24.8 per 1000. Christison found 27 and Heller 57 per 1000. The quantity lost in the twenty-four hours varies from 80 to about 400 grains (Frerichs, Gorup v. Besanez). The natural solid constituents of the urine are diminished in proportion to the obstruction in the kidneys. The excretion of urea falls to 100 or 200 grains (from 400 to 500 grains in health) and the inorganic salts are considerably lessened. Uric acid maintains about its usual quantity.

The *blood* becomes speedily deteriorated by the unnatural drain through the kidneys. It becomes more watery and poorer in albumen, while urea, uric acid, and the extractives are unduly accumulated in it. The blood-corpuscles diminish in number as the disease proceeds, and a generally anæmic appearance of the body is produced. Fibrine is usually in excess, and the blood displays a buffy coat. The fat and inorganic salts retain their usual proportion. Frerichs supplies the three following analyses of the blood in the early period of acute Bright's disease:

	I.	II.	III.
Specific gravity	1025	1022	1019
1000 parts of serum contained:			
Water	908.10	915.88	938.9
Solids	91.90	84.12	61.1
Albumen	81.40	72.00	51.7
Fat	1.42	1.53	9.4
Extractive matters and salts	9.09	10.59	

The pulse invariably shows high tension of the arterial system when examined by the finger or by the sphygmograph. In rare cases this may lead to a recognizable hypertrophy of the heart, even before the disease has passed into the subacute stage. Usually, however, cardiac hypertrophy is absent in acute Bright's disease.[1]

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[1] *See* Wagner, Ziems. Cyclop., 3d ed., p. 131. Also Riegel, Berl. klin. Wochen., 1882, No. 28, and Zietsch. für klin. Med., 1883, p. 260.

After the disease has persisted for a variable period of a few days to some weeks, it proceeds to one of three terminations, viz., recovery, death, or lapse into the chronic state.

When the case is about to terminate favorably, the urine increases in quantity to three or four pints daily; its density falls below the natural mean (1012–1008); and the blood, renal elements, and albumen gradually diminish and finally disappear from it. At the same time the skin becomes moist, and the serous effusions are reabsorbed. The rate of progress varies extremely. If albumen has totally left the urine in six weeks or two months, the recovery may be considered quick. The shortest period that I have known to elapse, from the first symptoms to complete reëstablishment of the normal state, has been ten days.

Some cases reach final recovery only after a protracted and interrupted convalescence of many months. The urine during this period continues abundant, of low density, occasionally of pink color from slight admixture of blood. The anasarca is also apt to recur and disappear, and recur again, perhaps several times, accompanied with febrile exacerbations of subacute character. In one such case observed by me the symptoms finally subsided in five months. The patient was seen ten months later, and the urine found perfectly free from albumen. In a second case, a slight admixture of blood continued, in diminishing quantity, for more than twelve months. In both these instances, and in a third similar to these, the characters of the urine were uniform; it was copious (three or four pints daily), of low density, slightly mixed with blood, slightly albuminous; the renal derivatives were devoid of fat, and, throughout the convalescence, comparatively scanty.

Not unfrequently, in the ordinary course of recovery from acute Bright's disease, the renal elements—both casts and epithelium—show slight signs of fatty changes. This circumstance is apt to embarrass the diagnosis, and lead to the suspicion of the existence of confirmed and chronic Bright's disease, if the case first come under observation in this stage. The doubt can only be solved by watching the progress of the case for a week or two.

But matters do not always take this favorable turn; and two new orders of symptoms arise, and bring life into imminent peril, or involve it in destruction. These are secondary inflammations of the serous membranes and the lungs, and uræmic intoxication.

Of the inflammatory complications, pericarditis is the most surely fatal, but it is rare. Pneumonia is more common; it breaks out without appreciable exciting cause, and usually runs a rapid course to a fatal end. Pleurisy and peritonitis are also not unfrequent, but greatly less to be feared. More or less bronchitis exists almost invariably. When the anasarca rises to an extreme degree, the integuments of the legs may inflame, and even mortify. These secondary inflammations are much more common in the later stages of chronic Bright's disease than in the acute disorder.

The uræmic phenomena are due to the retention in the blood of the excrementitious matters of the urine. They consist in a train of nervous symptoms—headache, vomiting, diarrhœa, convulsions, and coma—which are frequent incidents, and much to be feared in acute Bright's disease. They usually follow an excessive diminution or suppression of the urine from the increasing obstruction in the kidneys. It will be more convenient to postpone their consideration to a future section, when uræmia, in connection with Bright's disease generally, will be discussed.

Certain deviations from the usual course and symptoms are not unfrequently encountered. Although serous effusion generally first shows itself in the face, under the eyes, and then invades the trunk and extremities, it may begin elsewhere—in the feet, hands, or scrotum; or all parts of the body may swell up simultaneously. The effusion, too, may shift its place from time to time, or it may be poured out with disproportionate copiousness in certain localities (lung, pleura, submucous tissue of the glottis), and thereby determine sudden accession of alarming or fatal symptoms.

The anasarca commonly disappears some days or weeks, or even many months, before the albumen has vanished from the urine; but sometimes the converse is the case, especially in individuals of lax frames and anæmic tendency. When cases of this latter class come under observation for the first time after the urine has become free from albumen, they are very apt to mislead, and their true nature can only be recognized by a careful sifting of the patient's previous history.

Diagnosis.—The general symptoms, and the alterations of the urine, are so significant during the height of the attack, that the disorder can scarcely be confounded with any other. But when the pyretic stage is passed, and the case becomes protracted, there is often great difficulty in determining whether we have to deal with the declining periods of an acute and curable disorder, or with a disease which has already lapsed into the chronic and irremediable state, or with a disease which has been chronic from the first. Chronic Bright's disease is subject to occasional febrile recrudescences, which are deceptively like an attack of the acute disorder. The signs that the disease is acute and recent are: free presence of blood and renal epithelium in the urine, absence of fat in the discharged elements, absence of long-standing complications, such as hypertrophy of the left ventricle, phthisis, caries, necrosis, and joint disease. A careful consideration of the previous history and of the ostensible cause of the disorder is, also, of diagnostic importance. The less clearly a case can be traced to a definite exposure to cold, a bout of drinking, or to scarlet fever, or some other zymotic disease, the more reason is there, *pro tanto*, to fear that confirmed Bright's disease is established.

PROGNOSIS.—Precise data concerning the fatality of acute Bright's disease are wanting. A large majority of the cases undoubtedly recover. Frerichs reckons the recoveries as two-thirds of the individuals attacked. Probably this proportion is below the truth if the scarlatinal cases be included.

The signs of approaching resolution are: increased discharge of urine, diminished impregnation of it with blood and albumen, subsidence of the febrile phenomena, of the anasarca and serous effusions, and restoration of the cutaneous transpiration. At the same time, the countenance loses its stupid expression and its anæmic hue, and resumes its ordinary healthy aspect. The coexistence of all these signs leaves no doubt of advance toward a favorable issue: but the occurrence of some of them without the others must not lead to too sanguine expectations. The anasarca may disappear totally, and blood cease to tinge the urine; the quantity of the secretion may increase considerably, the pyrexia pass away, and the general well-being of the patient improve greatly; but if the urine continue to contain a considerable amount of albumen, there is strong reason to apprehend that the disease is lapsing into a chronic state, or that the amendment is but a temporary lull in the symptoms, to be followed at no distant period by an exacerbation, which shall prove more disastrous than the original attack. Recovery cannot in any case be considered complete, until the urine has become perfectly free from every trace of albumen.

If the urine become progressively scantier, of higher density, and more abundantly charged with albumen, tube-casts, and renal epithelium, the worst consequences are to be feared. The advent of inflammatory complications, of œdema of the lungs or glottis, and, above all, of decided signs of uræmic poisoning, are of equally evil augury, and leave but slender hopes of the final preservation of life.

The prognosis is decidedly more favorable in the aged than in the young. I have several times seen the disease in persons over sixty, and once in a man on the verge of eighty; but in most of them the disease proved mild, and in all of them it issued in recovery.

ETIOLOGY.—Acute Bright's disease, though not absolutely confined to any age, occurs, in the immense majority of cases, in childhood and youth. The individuals attacked are commonly of good previous health; in two instances, however, I have seen the disease complicated with acute pulmonary tuberculosis.

The exciting cause is usually some definite exposure to *cold* (a damp bed, wet clothes, lying or sleeping on the damp ground, sitting in a current of cold air, drinking cold water when in a state of perspiration), or a bout of drinking. A large proportion of the cases are sequelæ of scarlet fever, or (much more rarely) of some other zymotic disease. Albuminuria, and sometimes all the other phenomena of acute Bright's disease, have been described in cases of acute intestinal catarrh. Some cases are due to pregnancy.

TREATMENT.—If the case is seen at the time of invasion, the patient should be at once confined to bed, swathed in flannels, and made to lie between the blankets. The loins should be immediately cupped to eight or twelve ounces (in children to two or three ounces). After the abstraction of blood, a large linseed-meal poultice should be applied, hot, to the loins, and changed every three hours. A hot-water bath or a hot-air bath should be administered every evening, or every second evening. When no conveniences for a hot-water bath exist, an excellent substitute is found in the "blanket-bath." A large thick blanket is wrung as dry as possible out of boiling water, and wrapped round the body of the patient; the bedclothes are then heaped on. In twenty minutes or half an hour, the hot blanket is removed, and the surface quickly dried with a warm soft towel.

The bowels should be freely acted on every other morning by an active purge, such as the compound jalap powder. An endeavor should also be made to allay the fever and restore the action of the skin, by citrate of potash draughts, given every two hours, in effervescence, or a mixture of the liq. ammon. acet. in two or three drachm doses, with fifteen drops of tincture of henbane in an ounce of inf. lini. Dr. Barlow recommends tartar emetic in doses from ¼ to ⅛ of a grain. I have myself employed the same remedy with the best effects, every four hours. Dr. Johnson also speaks highly of antimonial wine, sometimes combined with Dover's powder.

The diet should be composed of light farinaceous substances, with milk, beef-tea, and broths. Flesh meat in any form is objectionable in the early stage.

The abstraction of blood must be cautiously practised, on account of the tendency to anæmia in the later periods of the attack; and if the patient's health is broken by previous disease, or is constitutionally weak, even local depletion is better omitted. If severe headache, coma, or convulsions occur, the cupping may be repeated. In very threatening, sthenic cases, where the fever runs high, venesection may be practised.

When the fever has abated, and the anasarca is yielding, the more active measures should be discontinued, or pursued in a less active manner; but the efforts to restore and maintain the action of the skin should be persevered in. In the later periods, when convalescence has been fairly established, preparations of iron should be substituted for the alkaline and diaphoretic remedies. It is always well to begin with small doses, and to feel one's way. A too early resort to ferruginous preparations may be followed by a return of the acute symptoms. When iron is tolerated, it acts with great benefit, and hastens in a marked manner the disappearance of blood and albumen from the urine. My experience agrees with that of Dr. Parkes, that gallic acid exercises no beneficial influence in the acute disorder.

The use of mercury is objectionable, on account of the extreme susceptibility of patients suffering from Bright's disease to the physiological effects of the drug. Severe salivation has been known to follow very small doses. In one of my patients two grains of blue pill, administered with extract of colocynth on two alternate mornings, produced profuse ptyalism.

The obstinate vomiting which occasionally prevails, may be combated with creasote, or small doses of chloroform, given in iced solutions. A careful revision of the diet should also be made. The gastric symptoms are sometimes due to direct sympathy with the renal irritation, and sometimes to genuine uræmic poisoning. The treatment of uræmia will be considered in a separate section.

The secondary thoracic inflammations present great difficulty in their management; they commonly set in when the patient is no longer in a fit state to bear the ordinary antiphlogistic means; and they run their course with unusual severity and rapidity. Counter-irritants and revulsives may, however, be energetically employed. Cantharides and turpentine should be avoided, from their special irritating effect on the kidneys; but hot-water applications, mustard poultices, and chloroform epithems may be applied locally over the chest, and to more distant parts—the calves of the legs, the feet, etc. Dry cupping over the chest is also a safe and sometimes valuable remedy.

When a favorable issue has been obtained, unusual care is required to guard against relapses, to which the patients continue liable for a considerable period. The slightest exposure is sometimes sufficient to reawaken the pyrexia, and to cause the reappearance of albumen and blood in the urine. A complete suit of flannels is essential; and, as a rule, the convalescent should not be permitted to leave his room until the albumen has disappeared from the urine. When that comes to pass (or before, if the case prove very lingering), change of air to a warm sheltered locality is likely to be highly beneficial, and to hasten the restoration of the impoverished blood.

Objections have been made, on theoretical grounds, to the use of the saline diuretics (acetate and citrate of potash) in acute Bright's disease. Experience has proved, however, that they may be employed with great advantage. They become changed in the *primæ viæ* into alkaline carbonates, and these diminish the acidity of the urine, and render it more bland as it percolates the renal substance. In a disease which tends to spontaneous recovery under simple hygienic and prophylactic treatment, it is necessarily a matter of extreme difficulty to bring home the evidence of the curative power of drugs; but in a considerable number of cases of acute Bright's disease, coming under treatment early, I have obtained almost invariably the best results by the free administration of citrate of potash. And in no instance where the urine has been rendered alkaline in the first week of the complaint, have I observed the more severe uræmic symptoms, or secondary inflammations. In the later periods, when the fever has altogether subdivided, while the urine still continues bloody and albuminous, the same medicament has not proved of any service in my hands.

Digitalis and broom-tops may be used freely in any stage to combat the dropsy. Dr. Christison recommends a combination of digitalis and bitartrate of potash as superior to either remedy given singly. "The former was usually given in the dose of one or two grains of the powder, in the form of a pill, three times a day, or in the dose of ten, fifteen, or twenty minims of the tincture, three times daily in a little distilled water of cinnamon or cassia. The cream of tartar was administered thrice a day in the quantity of a drachm and a half, or two drachms, with about five ounces of water. Diuresis may generally be induced by such means in the course of three or four days, sometimes sooner—seldom, however, if delayed beyond the seventh day."

Dickinson lays considerable stress on the desirability of encouraging the patient to drink freely of water, with a view of facilitating the elimination of the urinary solids by the kidneys, and thus diminishing the risk of uræmic intoxication.

Hamburger speaks strongly in favor of quinine in scarlatinal dropsy, after the pyrexia has abated. He gives to children 1½ or 2 grains, and to adults 3 to 4 grains, twice a day. Of 47 severe cases thus treated he obtained amendment in 44, either immediately or in a few days. ("Prag. Vierteljahrsch," 1861.)

CHAPTER IV.

CHRONIC BRIGHT'S DISEASE.

ANATOMICAL CHANGES IN THE KIDNEYS.

THE kidneys of persons dying of chronic Bright's disease present three chief types of alteration, viz.:

TYPE I.—*Kidney smooth, white*, and enlarged; in extreme cases, rarely met with, kidney atrophic (chronic nephritis).

TYPE II.—*Kidney granular, brownish, or red*, and contracted (cirrhotic kidney).

TYPE III.—*Kidney lardaceous or waxy* (so-called *amyloid* degeneration).

The special clinical history pertaining to each of these anatomical types has not been made out with sufficient precision to enable them to be invariably recognized during life; but much light has, in recent years, been thrown on the subject, enough to permit a synopsis of the symptoms, and conditions of origin, of the three types to be presented.

These types are not always found simple and unmixed. On the contrary, the main type of alteration present in any case is often complicated by superadded changes belonging to another type. Thus the smooth white kidney becomes not unfrequently affected with waxy degeneration or with interstitial growth; and the granular and the waxy kidney are each liable to inflammatory attacks, which bring about changes belonging to the first type. In this way a complex anatomical state is produced which is associated with a complex clinical history.

1. SMOOTH WHITE KIDNEY.

(*Chronic Nephritis.*)

The structural changes in the smooth white kidney are similar in kind to those already described as pertaining to acute Bright's disease, but advanced to a further stage; the surface continues perfectly smooth; the organ is considerably enlarged; the capsule is thin and easily stripped off. Conspicuous stellate patches of bloodvessels are seen on the white or mottled surface. On section the cortical substance is seen to be greatly increased; its color is ivory-white, or (in cases of fatty transformation) yellowish. The cones retain their usual color, but they appear conspicuously red from contrast with the abnormal whiteness of the cortex.

The microscopic changes, as described by Dickinson and Grainger Stewart, are essentially confined to the uriniferous tubes. The epithelial lining of the tube is enormously increased in quantity, and the tubes are thereby distended and enlarged. The cells are swollen, generally opaque and granular, and often largely charged with oily particles. In the pure form the intertubular tissue is not altered, but commonly it shows a little increase. In the progress of the disease a number of the distended tubes and their contents are broken up into a granular débris, and afterwards absorbed. Transparent fibrinous effusion and blood are sometimes seen within the tubules. The Malpighian corpuscles are either of their natural size or only slightly enlarged, and their capsules are thin, as in the natural kidney. The cones undergo changes of a corresponding character with those of the cortex, but less developed; and fibrinous casts are found occupying the interior of the straight tubes.

The large smooth kidney generally remains large and smooth to the last; but, sometimes, if the patient survive sufficiently long, the enlargement gives place to a progressive dwindling; and in very extreme cases, the kidney may be reduced to a weight of only an ounce, or even less. This dwindling is, however, not an invariable event, even when the patient survives for some years. Dr. Wilks relates the following case in point:

> "A young woman, æt. 23, had scarlatina *three* years before death. There was very slight eruption; dropsy soon followed, which lasted a year. Then the patient was slightly better, but remained an invalid, with œdema of the legs, until the last five months, when very extensive and general dropsy came on and persisted. The urine was then scanty, dark, and contained exudative deposit. She had three epileptiform fits, and death subsequently ensued from pleuritis and pericarditis. Lungs were found very œdematous. The aorta and arteries were covered with an atheromatous deposit; and the *kidneys* were large and white, with an abundance of deposit, much of which had undergone a fatty change." ("Guy's Hosp. Rep.," 2d series, vol. viii. p. 243.)

When the smooth kidney becomes atrophied, the capsule is somewhat thickened, and disposed to adhere to the renal surface, and slight superficial depressions make their appearance, giving the organ a slightly granular character.

This atrophic condition seems to be brought about by a destruction and gradual absorption of the distended tubules and their epithelial contents. The cortical substance is thus progressively consumed while the pyramidal portions retain their natural dimensions. The bloodvessels are found much thickened, and, according to Grainger Stewart, there is a relative increase of the fibrous stroma, but by no means to so great an extent as in the cirrhotic kidney.

The large white kidney is not unfrequently greatly infiltrated with fat. Oily particles are found in great numbers in the substance of the epithelium and lying free in the tubules. It constitutes one form of the "fatty kidney." This change sometimes begins at an early stage of the disease, but it only reaches an extreme degree in long-standing cases. Fatty transformation is much more frequent when the disease has arisen from cold than when it has followed scarlatina.

SYNOPSIS OF SYMPTOMS AND CONDITIONS OF ORIGIN.—The smooth kidney is found in those cases in which chronic Bright's disease has followed on the acute disorder. The invasion of the disease has been sudden, and it can usually be traced to some definite exciting cause, either cold or scarlatina. I have also seen the large white kidney in chronic Bright's disease following repeated pregnancies, and in a case arising in the course of phthisis.

The average age of 106 cases of smooth large kidney, examined by Dickinson, was 28.2 years; in 11 cases of smooth dwindled kidney the average age was 43.6 years; whereas in 250 cases of granular kidney the average age was 50.2 years.

Serous effusion is an almost invariable coincidence; the body is commonly bloated with dropsy; the face pale and puffy, and the cutaneous surface conspicuously white, smooth and glossy. There is also a markedly greater tendency to secondary inflammations, and to uræmic accidents, than in granular kidney, but less to valvular heart disease and hypertrophy of the left ventricle.

The urine is generally scanty. Its specific gravity is either normal, or somewhat raised above the usual average. It is pale and cloudy, but sometimes smoky and tinged with blood. On standing, it deposits a quantity of amorphous renal débris and casts of tubes. The casts are of various character, "epithelial," "granular," "fatty," and "hyaline." Cells having the appearance of pus-corpuscles are common towards the later periods.

The disease is of shorter duration than the granular kidney. In fatal cases the ordinary duration of the disease is under six months. Temporary recoveries and relapses are frequent. Permanent recovery may be hoped for even after the lapse of a year or more. In these protracted cases the albuminuria continues long after the dropsical symptoms have passed away. I have known cases in which abundant albuminuria has persisted for more than a year after all other symptoms of disease had ceased. At length the albuminuria has gradually disappeared, and the reality as well as the appearance of health has been established.

Quite exceptionally the disease may be protracted for several years. Dr. Johnson records an instance which endured for ten years, with good preservation of health for a portion of that period. Nine years before death, the urinary deposit clearly indicated fatty degeneration of the kidneys. After death, the kidneys were found dwindled to an ounce and three-quarters for the pair.[1]

2. GRANULAR CONTRACTING KIDNEY.

(*Cirrhotic Kidney.*)

The gland is diminished in size and reduced in weight. In extreme cases the weight of the kidney is reduced to two or three ounces, or less. Its surface is rough, and beset with numerous rounded elevations, varying from the size of a pin's head to a hemp-seed, or even a small pea. The capsule is opaque, thickened, and adherent to the subjacent surface, so that it cannot be peeled off without tearing the glandular structure. In certain spots the capsule sinks into the substance of the cortex, and divides the kidney irregularly—giving it a lobular appearance. On section, the cortex is manifestly atrophied, as compared with the cones, and forms a thin rim of only a line, or less, in thickness around the bases of the pyramids. It has a red, or brownish-red color, and a coarse granular texture. The entire organ is tough and resistant. In the granular kidney produced by gout there may be seen in the pyramidal portion longitudinal white or yellowish streaks, caused by a deposit of urate of soda.

When a thin section of a granular kidney is placed under the microscope, the secreting tissue is found to have undergone extensive destruction. The Malpighian bodies are shrunk to half their size, and unnaturally crowded together. Their vascular tufts are embraced in a fibrous and granular investment, and, in extreme instances, compressed into an impermeable knot at the bottom of their capsules.

The investment is formed by an increase of the fibrous tissue surrounding the glomerulus, or by a swelling and fibrous thickening of Bowman's capsule and its lining epithelium. The vascular tuft itself is not unfrequently transformed into a fibrous mass, and it may also undergo a colloid change.

The uriniferous tubes are altered in various ways, and to various degrees. Some are denuded of epithelium and reduced to mere tubular threads; others, equally denuded, contain glassy fibrinous cylinders; while others are crammed with broken-up epithelium. Many of the convoluted tubes are seen to be much diminished in size and lined by a layer of cubical epithelium instead of the large granular cells commonly found there.

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[1] Med.-Chir. Trans., vol. xlii p 160

Oil is found not unfrequently both in the fibrinous exudation and in the disintegrated epithelium, but not so commonly nor so abundantly as in the smooth kidney. Amid tubes changed to this degree, are found others not much altered, and lined with their proper and healthy epithelial investment. The basement membrane of the tubes is thickened. Between the wasted structures lies a large quantity of adventitious connective tissue, which gives the organ its peculiar toughness.

The arteries of the kidney show considerable changes. They may show thickening of all their coats. Dr. George Johnson first pointed out the great increase in the muscular coat, and although his position has been attacked by some, it has been abundantly confirmed by other observers. Dr. Klein has observed what is probably the early stage of this change in the scarlatinal kidney, where he found proliferation of the muscle nuclei in the walls of the arterioles. The adventitia frequently shows fibrous thickening, which merges into the general connective tissue of the kidney, while the intima very often is the seat of chronic endarteritis, which may considerably diminish the lumen of the vessel.

It is only rarely that the granular kidney is encountered in the early stage of its development. When such is the case, thickening of the capsule and slight granulation of the surface are found to precede contraction, so that the organ at this period preserves its normal volume. The granular kidney, in the contracted state, allows injections to penetrate imperfectly. Dickinson found that when a stream of warm water was propelled through the bloodvessels, a very considerably less quantity passed in a given time than through a healthy kidney—less also than through the large smooth kidney—showing that the permeability of the gland to the blood-stream was greatly lessened. In the healthy kidney, the mean discharge through the renal veins in ten minutes was 119 ounces; in the large smooth kidney, 90 ounces; and in the granular contracted kidney, 25 ounces. This diminished permeability is not wholly due to the cirrhotic state of the intertubular matrix, but partly to thickening of the walls of the minute arteries of the kidney, as has been pointed out by Dr. G. Johnson. ("Brit. Med. Journ.," April, 1870.)

Thoma has obtained results similar to those of Dr. Dickinson, using as the injecting fluid either defibrinated ox-blood or gelatine solutions. He lays stress on the endarteritis as a cause of obstruction to the blood flow, but also shows that the obliteration of the glomeruli may take part in this, and that the arteries of the kidney allow of more easy transudation through their walls. The influence of the destruction of small vessels is to some extent counteracted by anastomoses which are set up. Thus, when the vascular tuft of the glomerulus is destroyed, a direct communication is established between the afferent and efferent vessels; when the glomerulus becomes cystic, a system of capillaries running around the cyst-wall again establishes communication between the afferent and efferent vessels; in addition, the normal anastomoses between the branches of the renal and neighboring arteries become dilated, and carry off a larger amount of blood than usual.

Of the intimate nature of the process which finally brings about this state of the kidney, there are conflicting opinions. Dr. Johnson believes that the mischief begins in the epithelial cells, and that the fibrous tissue is the remnant of atrophied tubes. Dickinson and Grainger Stewart describe the process as originating in the intertubular matrix, and as consisting essentially in an enormous hypertrophy of the fibrous stroma of the organ. By the pressure and contraction of this fibrous material the uriniferous tubes and Malpighian corpuscles are extensively destroyed, and the size of the kidney is progressively reduced. According to Dickinson, the fibrous growth begins beneath the capsule, and then penetrates into the interior of the cortex. The points where the fibrous processes penetrate are depressed, and when these are numerous and distributed with tolerable regularity, the appearance of superficial granulation is produced. The disease thus travels from the surface towards the central parts, and eventually involves the pyramids. It must be admitted, however, that in many specimens the fibrous tissue can be seen grouped round the glomeruli and the arteries, and not proceeding from the capsule.

Sir William Gull and Dr. Sutton propound a different view of the pathology of granular kidney. They assert that the morbid process consists in the deposit of a "hyalin-fibroid" material in the fibrous coats of the small arteries and capillaries. This morbid change is attended with atrophy of the subjacent tissues: it is not confined to the kidneys, but may prevail extensively throughout the *area vasculosa.* Although it generally begins in the kidneys, there is evidence of its beginning primarily in other organs. A brief account of these views is given further on in treating of the connection of Bright's disease with diseases of the heart and vascular system.

The development of cysts is very common in the granular kidney. They vary in size from a pin's head to a pea or a hazel-nut; but many are so minute that they can only be detected by the microscope, not being larger than the width of the uriniferous tubes. Mr. Simon believes that they are formed by an immense dilatation of epithelial cells—a development that seems incredible, considering the fragility of the outer portions of these cells; and one that would be, so far as I know, without parallel in histogenesis. A better sustained, and more commonly accepted, view is, that they are produced by obstruction of the uriniferous tubes with exudation, at intervals, or by compression of their walls at interrupted spots by the contracting adventitious tissue. The spaces thus enclosed become distended with a serous fluid, and are sometimes found lined with an epithelial layer. Their contents are not urinous, but consist of an albuminous saline solution or of a colloid mass. In the cones they are sometimes elongated and placed end to end like a string of sausages (Dickinson). (*See* Cysts and Cystic Degeneration in the Kidneys.)

SYNOPSIS OF SYMPTOMS AND CONDITIONS OF ORIGIN.—The granular kidney is found in the vast majority of those cases of Bright's disease which are chronic from the beginning—those which commence insidiously, without definite exciting cause. Dropsy is altogether absent in a considerable proportion (perhaps in a quarter—according to some, in one-half) of the cases, and when present, it is commonly slight and limited to œdema of the ankles and legs, or a puffiness under the eyes. It often disappears for a while, and returns again.

The disease may run a latent course for months and years. A deep constitutional cachexy is associated with it in a large proportion of cases. The subjects of it are more advanced in years than those of the smooth large kidney (*see* p. 398).[1] The cutaneous surface, though pale and anæmic, has not the conspicuous whiteness of the preceding type, and the features are often pinched and sallow. With this type of Bright's disease are especially associated hypertrophy of the left ventricle and certain changes in the small arteries and in the retina, which will be more fully noticed in a future page. (*See* Complications.)

The urine is copious—three or four pints a day—and of low specific gravity; the quantity of albumen is comparatively small; in rare cases it may even be temporarily absent from the urine. Toward the termination of the disease, however, the urine becomes scanty, or even suppressed. The deposit is slight, composed of hyaline and granular casts, with very slight admixture of epithelium, not often fatty: the deposit is often so scanty that it may escape detection, or there may really be none. As a rule, blood is absent.

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[1] Young persons and even children are, however, sometimes the victims of this type of Bright's disease. I saw a remarkable example with Dr. Barlow, in a child of 5 years, of which Dr. Barlow has given an elaborate account in the Lancet for Aug. 1, 1874.

The common predisposing causes are habitual intemperance, gout, lead poisoning, repeated exposure to cold, and extensively distributed fatty degeneration of the tissues.

As these two types—the smooth large white kidney, and the granular red contracting kidney—constitute the vast majority of cases of Bright's disease, the question of the oneness or multiformity of Bright's disease has mostly been limited to the inquiry—whether the latter is the ultimate stage of the former, or whether the two are distinct from first to last.

Reinhardt and Frerichs believe that the large white kidney will, if the patient survive, eventually become granular, red, and contracted. This view has obtained numerous supporters in Germany, and in Rosenstein's work it is adopted without discussion. In this country the opposite view has steadily gained ground; and the evidence brought forward by Johnson, Wilks, and Dickinson appears to place the matter beyond reasonable doubt. In Germany, too, several recent writers, amongst whom are Bartels, Senator, and Leyden, have adopted this view.

Johnson found that out of 26 fatal cases of enlarged kidney, observed by himself, there was dropsy in 24, or 92 per cent.; whereas in 33 fatal cases of contracted kidney, there had been dropsy only in 14, or 42 per cent. He pertinently observes: "If all the contracted Bright's kidneys have passed through a previous stage of enlargement, it is difficult to understand how it can happen that the majority of those patients who have reached the final stage of renal degeneration should escape the dropsy which, in a greater or less degree, troubles nearly all those who die in what is assumed to be an earlier stage of the same disease."[1]

Convulsions and secondary inflammations (pneumonia and peritonitis) are more frequent with the smooth kidney; whereas hypertrophy of the left ventricle, atheroma, and apoplexy are more frequent with the granular kidney.

It must, of course, be borne in mind that it is not now disputed that the large white kidney does sometimes suffer atrophic changes, and that in exceptional cases it may at length dwindle to very small dimensions. Johnson and Dickinson and Grainger Stewart adduce several examples of such a change, but they insist that even in its further stage of contraction the smooth white kidney is still distinguishable from the granular red kidney. The change of the large white kidney to the atrophic form is oftener accompanied by a similar change in the clinical features; the symptoms then approach in character those of the small red granular kidney.

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[1] Med.-Chir. Trans., vol. xlii. p. 156.

The symptoms of the two varieties of Bright's disease mentioned above are not yet sufficiently worked out to enable us always to prognosticate from the clinical signs the exact lesion to be found after death. Various "mixed" forms are occasionally met with. The small granular kidney is subject to attacks of acute inflammation and congestion, and under the influence of these may show many of the changes of the large white kidney. Leyden has called attention to cases where the symptoms are entirely those of the small red granular kidney, yet after death the kidneys are found large and pale. The microscope in such cases shows excess of interstitial tissue, as in the contracted form, but combined, it may be, with parenchymatous changes.

3. Lardaceous or Waxy Kidney.

(*So-called Amyloid Degeneration of Virchow; Depurative disease of Dickinson.*)

Most pathologists now distinguish the waxy or lardaceous kidney from the preceding varieties of Bright's kidney.

Externally, the waxy kidney is smooth, or sometimes slightly roughened; the capsule peels off readily. The organ is usually enlarged, sometimes, however, diminished in size. On section the appearance is characteristic. The cortex is bloodless, of a white or yellowish color, with a waxy, smooth, translucent appearance, resembling bacon-rind. The organ is conspicuously tough and hard. On the smooth cut surface, little appearance of the natural secreting structure is seen, but it is dotted over with bright glancing points: these are the changed Malpighian bodies. The cones appear unnaturally red and distinct. This description answers only to extreme degrees; in slighter cases the change can only be detected by the use of reagents.[1] When a thin section thus prepared is examined under the microscope, the waxy change is seen to affect mainly (sometimes exclusively) the bloodvessels. The Malpighian corpuscles are the parts earliest attacked. Without reagents they appear as shining particles with thickened capsules; the vascular tufts are greatly swollen and give the characteristic reaction. In advanced cases, the vasa afferentia, with the arteries and capillary network of the cortex, and even the vessels of the pyramids, are similarly changed. The lumen of the bloodvessels is much diminished by the deposit.

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[1] For naked eye purposes the liquor iodi of the British Pharmacopœia, diluted with water until it has the color of brown sherry, is recommended as a convenient test for the waxy degeneration (Dickinson). The iodine imparts to the affected portions a mahogany-brown color, whereas the parts not affected take a merely yellowish tinge. For microscopic purposes methyl-violet is the best test. This reagent colors the amyloid parts red, and the healthy parts blue.

Dr. Dickinson has described a morbidly rigid character of the renal tubes, when the kidney is examined in the earlier stages. Later on, the epithelial cells of the uriniferous tubes are commonly withered, often infiltrated with fatty molecules; only rarely are they the seat of the waxy change, but the basement membrane of the tubes is frequently affected.[1] Hyaline waxy casts exist in some of the tubules. The stroma of the gland is usually increased in quantity and in some cases to an extreme degree. The kidney, then, may be contracted and may resemble in microscopic characters the small red granular kidney.

The liver and spleen are usually enlarged, and in a lardaceous state, when the kidneys are so affected. Of 77 cases collected by Rosenstein, the three organs together were affected in 48; the spleen and kidneys in 20; the liver and kidneys in 4; and the kidneys alone in five cases.

The chemical nature of the waxy material has only recently been investigated. Virchow concluded from the violet color produced by iodine and sulphuric acid that it belonged to the same group as starch and cellulose, which likewise yield a violet tint with the same reagents. But the ultimate analyses of C. Schmidt and Kekulé show that it contains nitrogen—and indeed as much as 15 per cent., or almost exactly the same proportion as the protein compounds. Neither of the chemists named could produce a particle of sugar from it by boiling with dilute sulphuric acid. It further resembled albuminous compounds, in yielding a violet color with the cupropotassic solution, in dissolving completely in dilute caustic potash, and in being precipitated from this solution in white flocks by acids.[2] The proportion per cent. of carbon, hydrogen, and nitrogen, found by Kekulé, in purified waxy matter, from an exquisite specimen of lardaceous spleen, was: C 53.58; H 7.00; N 15.4—which corresponds closely with the percentage of the same elements in albumen. Dickinson considers it a variety of fibrine—but differing from ordinary fibrine in containing about one-fourth less alkali and a somewhat larger proportion of earthy salts. To call it "amyloid" is simply a misnomer, and an unfortunate one, because it leads to confused notions as to the existence of some connection between waxy degeneration and the (genuine) amyloid substance found in the healthy liver.

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[1] Dr. G. Stewart states that, occasionally, epithelial cells are found on the casts in the urinary deposit, which exhibit the peculiar reaction of lardaceous matter.

[2] Friedreich says that the "amyloid reaction" (with iodine and sulphuric acid) was obtained by him in perfection, with decolorized fibrine from an old hæmatocele.

SYNOPSIS OF SYMPTOMS AND CONDITIONS OF ORIGIN.—Waxy degeneration of the kidneys always comes on insidiously and in cachectic persons debilitated by some preëxisting wasting disease. In 145 instances collected by Fehr it coexisted with the following disorders:[1]

Pulmonary tubercle	43	cases.
(complicated with caries in 5 cases, with other diseases in 6 cases.)		
Syphilis	34	"
Caries with struma	26	"
Empyema with fistula	4	"
Dilated bronchi with chronic bronchitis	3	"
Chronic alcoholism	5	"
Chronic articular rheumatism	2	"
Cancer (generally of the uterus)	3	"
Ague.	4	"
Chronic nephritis with hydronephrosis	3	"
Chronic peritonitis, scarlatina, variola, cirrhosis of the liver, ovarian tumor, urethral fistula with stricture . . each	1	"
Atonic ulcers of the foot	3	"
Without appreciable cause	9	"

It has also been found in association with gout, rickets, various abdominal tumors, and mercurial intoxication.

The aspect of patients with waxy kidneys is pale and cachectic, and the course of the disease is essentially chronic. Dropsy is present in the majority of the cases (in 98 out of 152 collected by Fehr); in some it is abundant and general, in others slight and partial. Uræmic symptoms are strikingly infrequent.

The urine in the earlier stages of the disease is markedly abundant—60, 100, or even 200 ounces per day—and Dr. Grainger Stewart has pointed out the important fact that this polyuria is a marked feature even before the urine becomes albuminous, and that this supplies a warning of the approaching advent of this form of Bright's disease. The quantity of albumen is at first small, but as the disease advances the urine becomes scantier, the proportion of albumen very great, and the specific gravity high. The color of the urine is commonly pale, and it allows only a very slight deposit to subside. This consists of casts and atrophied renal cells, which are sometimes fatty. Cells resembling those of pus are occasionally found either separate or aggregated round a cast. The tube-casts are usually hyaline, and they do not yield a brown coloration with iodine. Epithelial casts are also sometimes seen.

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[1] A. Fehr. Ueber die Amyloide Degeneration, insbesondere der Nieren. Bern, 1867.

[2] Litten has described cases in which the amyloid change had proceeded to an advanced stage without albumen appearing in the urine (Berl. klin. Wochensch., 1878, Nos. 22 and 23). Strauss has found that in such cases the arteriæ rectæ are the parts most affected. (*See* Cornil and Brault, loc. cit.)

Münch detected "corpora amylacea" in the urine of a man with lardaceous kidney; they were constantly present, and were colored violet by iodine and sulphuric acid.[1]

The diagnosis of waxy kidneys rests partly on the coincidence of a pale abundant albuminous urine with dropsy, but chiefly on the coexistence or preëxistence of one of the wasting diseases of which waxy kidneys are known to be a frequent complication, namely, phthisis, caries, long-continued suppuration, and constitutional syphilis.

GENERAL COURSE AND SYMPTOMS OF CHRONIC BRIGHT'S DISEASE.

Chronic Bright's disease, in the great majority of instances, begins slowly, imperceptibly. It is rarely detected until it has already existed some months—it may be, years. The attention of the patient is at length awakened by the gradual failure of his strength, the increasing pallor or sallowness of his complexion, and his disinclination to exertion; perhaps his suspicions are aroused by a little puffiness under the eyes, a slight swelling of the ankles at night, unusually frequent calls to void urine, or shortness of breath.

In other cases these premonitions are altogether wanting, or perhaps they pass unheeded. The fatal disorganization in the kidneys proceeds silently, amid apparent health, and then suddenly declares itself by a cerebral hemorrhage, a fit of convulsions, rapid coma, amaurosis, pulmonary œdema, or a violent inflammation.

Or, again, the disease creeps on stealthily in the wake of some preëxisting chronic disorder—phthisis, caries, necrosis, joint disease, constitutional syphilis, gout, chronic alcoholism, or exhausting suppuration.

Or, it may be a continuation or sequela of acute Bright's disease.

Lastly, the disease may lie concealed for an undetermined period, and then reveal itself after exposure to cold or a fit of intoxication, in the guise of an acute attack—with rapid general anasarca and scanty sanguineous urine.

The principal symptoms of the disease are: albuminous urine with deposits of tube-casts and renal epithelium; dryness of the skin; frequent micturition, especially at night; dropsical effusions into the subcutaneous cellular tissue, serous cavities, or pulmonary substance; derangements of digestion; progressive hydræmia; uræmic phenomena (headache, amblyopia, convulsions, coma, vomiting, and diarrhœa); hypertrophy of the left ventricle; secondary inflammation of the parenchymatous organs and serous membranes.

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[1] Cited by Parkes. Composition of the Urine, p. 394.

Few cases present the whole of these symptoms; and many present only two or three of them. The alterations in the composition of the urine are the most invariable; they are also the earliest and most distinctive symptoms; next follow, in the order of constancy, the deterioration of the blood, the dropsical symptoms, and lastly the uræmic and inflammatory incidents.

The disease usually pursues an interrupted course. It is subject to exacerbations from time to time, with intervals of quiescence. The exacerbations are generally occasioned by exposure to cold, or some imprudence in diet or regimen; sometimes no cause can be assigned for their occurrence. They are marked by pyrexia; and resemble, often closely, an attack of acute Bright's disease. The intervals of quiescence may be longer or shorter, some weeks or months, or a few years; the remission of the symptoms is commonly only partial—the main features of the disease persisting, though in diminished prominence. Sometimes, however, the remission is almost complete, and there remains little except the albuminous state of the urine to attest the existence of renal mischief. Nay, even this may, in very exceptional cases, be absent, and the nature of the case be first revealed at the autopsy.

After each exacerbation it is commonly pretty evident that the disease has taken a step in advance, and assumed a fuller development; and that, probably, an additional portion of the kidney, hitherto spared or only slightly affected, has been disabled.

But whether it thus proceed *per saltum* or more continuously, the kidneys are at length so deeply injured, and their depurative functions so far abrogated, that life falls a forfeit.

The immediate cause of dissolution is various. Sometimes the sufferer passes peaceably away exhausted by anæmia, burdensome anasarca, and defective digestion of food. More frequently the final scene is tumultuous. Two of the cases to be hereafter related, terminated amid a pyrexial exacerbation, with formation of clots in the heart. About one-third of the subjects of chronic Bright's disease perish by uræmic poisoning, either in the form of coma and convulsions or irrepressible vomiting and diarrhœa. A considerable number die from the dangerous situation, or intensity, of the dropsical effusion—as when the lungs or glottis are invaded; or death comes from hydrothorax, or from gangrenous erysipelas set up in the tense hydropic integuments of the thighs, legs, or genitals. About one-fifth die by secondary pneumonia, pericarditis, or double pleurisy. The remainder are cut off by less closely connected complications—apoplexy, cirrhosis, phthisis, intestinal ulcerations, etc.

From the difficulty of assigning the exact date of invasion, the *duration* of the disease can only be approximately ascertained. Enough is, however, known, to show that it varies within very wide limits. The usual period is from two to three years; but cases may end in six months, or be protracted for four or five years. Exceptional instances have been recorded, in which patients have survived 10 years (Johnson and Kussmaul), and even 15 (Bright) and 23 years (Oppolzer). The large white kidney is usually fatal much more quickly than the red contracted variety.

The following abstracts of cases will serve to exhibit the broad features of the disease, in its more familiar aspects; and prepare the way for a more detailed consideration of the symptoms and complications:

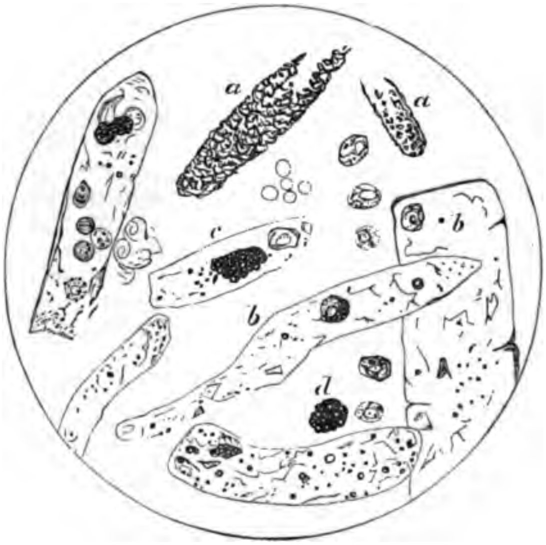
CASE 1. *Chronic Bright's disease, latent two years, without dropsy—fatty casts and cells in the urine. Death by uræmic convulsions.*—Mr. V., a solicitor, of temperate habits, æt. 50. Two and a half years ago, Mr. V. suffered from sciatica, for which he was under medical treatment. At that time a little albumen was discovered in the urine, but slight importance was attached thereto. Mr. V. speedily recovered from his sciatica, and continued in good health, attending to his business, until four months ago, when he became subject to shortness of breath and catarrhal symptoms. These were not severe enough to prevent the patient from pursuing his occupation, until the beginning of April, 1864, when I was requested to see the case with the late Mr. Mellor. The symptoms complained of were, shortness of breath on exertion, and failure of strength; there was not a particle of œdema (nor had there ever been any) nor ascites. The liver and spleen were not enlarged; there were no cardiac murmurs; but there existed slight præcordial bulging, and the heart's apex beat in the vertical line of the nipple. The shortness of breath evidently depended on pulmonary œdema. The countenance was pale and sallow, and the body spare, but not conspicuously emaciated. The urine was copious (three pints), of low density (1012), and highly albuminous (⅓); it deposited a not inconsiderable flour-like sediment, composed of casts and renal epithelia, many of which showed abundant signs of fatty changes (*see* Fig. 51). The casts were mostly medium sized; some were granular and opaque, as at *a*; others, in about equal numbers, were nearly hyaline, with only very faint markings, as at *b*. Withered epithelia studded some of the casts, or lay scattered free about the field. Botryoidal fat masses lay embedded in some of the casts; other casts were dotted over irregularly with oily particles. Some of the renal cells were similarly dotted in their interiors, while others were entirely changed into round agglomerations of fat molecules (granular corpuscles) (*c*, *d*). A few sparse blood-dsks were scattered about.

The previous history was singularly barren of etiological indications. The patient's mode of life had been strictly temperate; and there was no evidence of repeated exposure to cold, nor of gout. Father and mother died at the age of forty-five—the latter of consumption. He himself had enjoyed remarkably good health, until the invasion of his present complaint.

The treatment adopted was: dry-cupping the chest, warm bath every second day, flannel clothing, cod-liver oil, and iron. The dry-cupping removed the dyspnœa at once, and some general amendment took place in the course of the ensuing month.

This gentleman continued under observation until his death, which took place in three months. He improved for a while, and was able to go to Southport for a fortnight, where he derived considerable benefit. He considered himself so well on his return, that he believed a week or two would complete his recovery. He resumed his usual occupation, and, for a week or two, went daily to his office. But this truce was wholly deceptive; the condition of the urine never improved. It became progressively scantier in quantity—first it fell to 40 ounces, and then to 30 ounces, while the specific gravity continued to range from 1009 to 1011; and the deposit of casts became more and more opaque-granular, and less and less fatty. Emaciation also progressed, and the shortness of breath returned, and could no longer be kept under by dry-cupping. A persistent feverishness began to prevail; the nights were restless; but during the day the patient was dull, almost drowsy, and indifferent. Not a trace of œdema appeared throughout the complaint. The hypertrophy of the heart became progressively more conspicuous.

FIG. 51.



Casts and renal cells from the urine of Mr. V. *a a.* Granular opaque casts; *b b.* Hyaline casts; *c, d.* Fatty masses.

In the last fortnight of life, the urine became very scanty (still of low density), and was totally suppressed for twenty-four hours before death; vomiting recurred frequently, with utter loathing of food, and especially of animal flesh. The sight failed, and two days before death he became completely blind for more than half an hour. The restlessness increased, accompanied with wandering delirium, the tongue became dry, the indifference merged into drowsiness, and, after a fit of convulsions, he died.

The general course and symptoms clearly indicated a granular contracting kidney; but the friends would not permit a post-mortem examination.

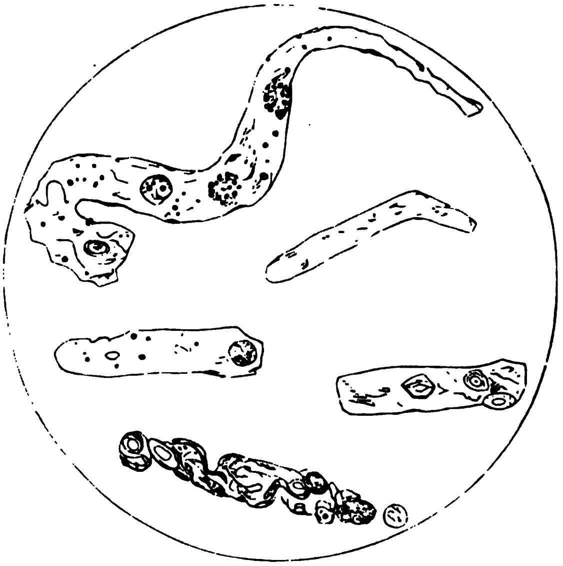
Case 2. *Chronic Bright's disease from intemperate habits — sudden anasarca after a wetting. Death from pericarditis. Granular contracted kidneys.*—W. M., a carter, æt. 40, of intemperate habits, was admitted into the Royal Infirmary, March 1, 1858, with general anasarca and ascites. He had followed his employment, and considered himself in good health, until three months back, when he got a severe wetting, and allowed his clothes to dry on him. Soon after followed lumbar pains and general swelling of the body. On admission there was œdema of the face, trunk, and extremities, and considerable ascites. The skin was dry; the urine, of low specific gravity, contained tube-casts, but no blood. After he had been in the house a fortnight, the urine became scanty, and intense pericarditis set in, which proved fatal on the fifth day. He died comatose with suppression of urine. At the autopsy the kidneys were found granular and greatly atrophied; scarcely any cortical substance remained. Abundance of fibrinous exudation existed in the pericardium; left ventricle immensely hypertrophied—the walls fully one inch thick; the valves were healthy.

The state of the organs after death, indicated that the disease had been really in existence for a much longer period than the few months during which symptoms had been noted by the patient.

Case 3. *Chronic Bright's disease from repeated pregnancies—recurrent uræmic convulsions. Granular contracted kidneys.*—Mrs. X., æt. 39, became pregnant of her sixth child in the autumn of 1862. About the third month, unusual frequency of micturition at night was observed, and soon after slight œdema of the face and legs. The urine was found to contain albumen. The fœtus was expelled without accident at the fifth month, and a few days after all the œdema disappeared; but the urine still continued albuminous. I first saw her about two months after the miscarriage. There was no œdema of any part. The urine was of low density, and moderately albuminous. The deposit contained a few transparent tube-casts, some of which showed slight evidences of fatty change; others were opaque, and studded with withered epithelia (*see* Fig. 52).

The patient continued under observation for above a year, and died, at length, comatose, after repeated attacks of convulsions. Each catamenial period was marked by great nervous excitement; and on several occasions convulsions took place at these periods, accompanied with temporary amaurosis. Severe headache was a very constant symptom, especially on the days preceding the catamenial periods. After death the kidneys were found granular and atrophied, and the left ventricle much enlarged.

FIG. 52.



Transparent and opaque casts from the urine of Mrs. X.

CASE 4. *Chronic Bright's disease from intemperance and exposure to cold—general dropsy, complicated with old chronic peritonitis. Death from syncope. Smooth white kidney, beginning to contract. Myriads of minute uric acid calculi in the infundibula.*—J. R., æt. 48, a French polisher, from Oldham, was admitted into the Royal Infirmary, April 4, 1864.

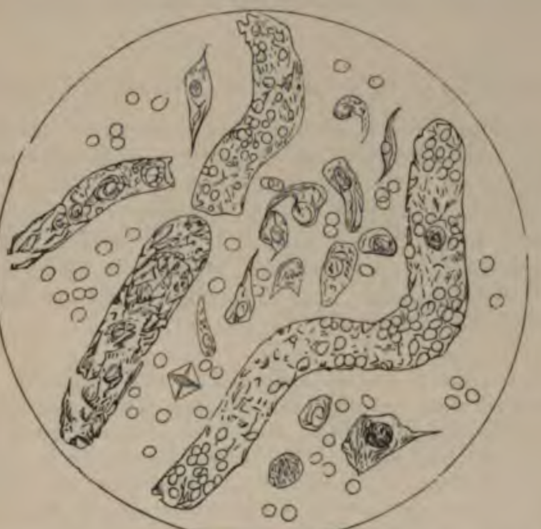
There was great ascites, tense œdema of the lower extremities, with an erysipelatous state of the integuments of the upper and inner parts of the thighs and scrotum; œdema also of the arms and back of hands. The heart was displaced upwards, and much enlarged; there were no cardiac murmurs. There was great emaciation, cough, purulent expectoration, and orthopnœa.

The urine was scanty, dark-colored from blood, highly albuminous; it let fall an abundant chocolate-colored deposit, composed of "blood-casts," "granular casts," and "epithelial casts," with abundance of free renal epithelium and free blood-disks. Mixed with these were a large number of irregularly tailed and spindle-shaped cells, evidently from the pelvis of the kidney (*see* Fig. 53).

The patient stated that he had been ailing twenty weeks; the symptoms had come on gradually. The swelling had first appeared in the belly, and the enlargement of the abdomen was still out of proportion to the general dropsy. His habits had been for years intemperate, and he was often exposed to chills, in suddenly passing from his warm work-shop to the cold open air. He had, however, been a healthy man, and had never lost a day's work until his present illness.

He went on, with little change in the general symptoms and urine, for twenty-five days, when he became feverish and delirious, apparently from cold, taken by imprudently exposing himself after a warm bath. He suddenly fell back dead on April 27th, as the nurse was shifting him for the purpose of making his bed.

FIG. 53.



Blood-casts, granular casts, blood-disks, tailed and irregular cells from the pelvis of the kidney—from the urine of J. R.

Autopsy, 24 hours after death. There was a good deal of anasarca of the lower limbs, forearms, and hands. An enormous quantity of serum escaped from the peritoneal cavity. The peritoneal membrane, in its entire extent, was thickened, or, rather, it was invested with a layer of thin adherent fragile false membrane of a pearly translucency, like the hyaline membrane of a hydatid sac. The intestines were sunk on the spine; there was no recent peritonitis. The liver was covered over with a rough layer of hyaline false membrane, which evidently embraced it tightly, and had caused it to shrink much below its natural bulk. On section it did not display a cirrhotic structure. The spleen was rather large; its capsule thick and opaque. The kidneys weighed together eleven ounces. They were firm, and their capsule smooth, but opaque and thickened. The capsule peeled off with only moderate ease, and tore the subjacent tissue a little. The surface of the gland was yellowish-white picked with dead white, like ivory. On section, the same appearance was seen to prevail throughout the cortical part. The cortex was, if anything, below its normal proportion. The pyramids were of a faint red color, not unnatural looking. The infundibula were somewhat dilated, and contained (in both kidneys) myriads of very minute yellow, uric acid calculi. These varied in size from a pin's head to an almost microscopic object; they were lumpy and irregular in shape. The papillæ were flattened, some of them almost obliterated. The kidneys were evidently of the "smooth white" species, beginning to pass into a state of contraction. The heart weighed 12½ ounces; the left ventricle was enormously hypertrophied; its walls seven-eighths of an inch thick. The right ventricle was also hypertrophied, and the tricuspid orifice somewhat patulous. All the valves were perfectly healthy. The lungs were strongly compressed, and partially airless and leathery from pleuritic effusion.

PARTICULARS OF SYMPTOMS AND COMPLICATIONS.

URINE.—The urine is albuminous to most varied degrees. It may become absolutely solid on boiling, or it may contain only the minutest traces of albumen, even in confirmed and fatally tending cases. The red granular kidney is accompanied by but a small amount of albumen in the urine, and even that may in rare cases be present only at certain times of the day.[1] The large white kidney, on the other hand, usually causes a large amount of albumen to be passed.

Absolute freedom from albumen, even for short intervals, is very rare; I am convinced, that a considerable number of the cases so reported, are examples of imperfect testing. But it must be admitted that chronic degeneration of the kidneys, not distinguishable from some forms of Bright's disease, does exist under certain circumstances, without albuminuria. The following example of scarlatinal dropsy, running a chronic course and ending fatally, without albuminuria, occurred in my practice:

J. K., æt. 8, was admitted into the Royal Infirmary, April, 1864, afflicted with general anasarca. She had had scarlet fever four months before, and during convalescence therefrom (in the third week), was suddenly seized with general swelling of the body, which has continued since. When admitted, she presented a perfect type of scarlatinal dropsy—universal and great anasarca, difficultly pitting on pressure. puffy pasty face, excessive pallor of the surface, shortness of breath. On examining the urine not a particle of albumen could be detected, nor any casts or other renal derivatives; it was scanty and high-colored. The skin was very dry, and a constant degree of feverishness existed.

She remained under observation until her death, four weeks after admission. The œdema remained stationary; the urine was repeatedly examined, but never found to contain albumen. The feverishness became more intense, the tongue became dry, and the breath very short; toward the close there was diarrhœa, which helped to carry her off.

Autopsy, 24 hours after death. Several deep and old tuberculous ulcers were found in the small *intestines*. A few nodules of tubercle, as big as peas, were grouped under the peritoneum, around the bases of these ulcers. The mesenteric glands were enlarged and tuberculous. There was no general tuberculosis of the peritoneum. The *lungs* contained a few old tubercles (of no great size) at the apices. The tuberculous masses were throughout old and inactive.

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[1] Dr. Mahomed maintains that in the pure red contracting kidney, no albumen is passed, and that it is only under the influence of an intercurrent affection of the renal epithelium that the albumen makes its appearance.

Both *pleuræ* contained a large quantity of fluid, and the lungs were much compressed thereby.

The *liver* was excessively bloodless. The *heart* was natural.

The *kidneys* were good examples of the "smooth white" Bright's kidney. They were slightly enlarged, and weighed together 7 ounces. The organs were limp, their surface pale and smooth; the capsule peeled off readily. The most curious thing about them was the existence of certain sharply outlined flat depressions, which differed from the remainder of the superficies. The surface generally was of a characteristic fawn-color, picked with dead white; but at these depressed spots the color was slate-gray, and contrasted markedly, by its blank, gray aspect, with the spotted appearance of the remainder. It was evident that atrophic changes were beginning to take place at these spots. On section, the kidneys presented the usual appearance of the "smooth white" kidney.

There was no information as to the state of the urine when the anasarca broke out; but for a month preceding death it was free from albumen, though the general symptoms, and the state of the kidneys after death, bore evidence of the existence of Bright's disease.[1]

The amount of albumen lost in twenty-four hours varies commonly from 45 to 300 grains. Dr. Parkes observed in one instance as much as 545 grains. The quantity is larger during digestion than during fasting; it may be twice as great. It rises and falls irregularly in the course of the disease—sometimes diminishing to a trace, and anon increasing to an intense impregnation.

The urine is generally pale, and not quite clear. It deposits, on standing, an amorphous whitish sediment, of renal epithelium and tube-casts. It sometimes contains blood—even in quantity—though generally only in microscopic proportion. When there is intercurrent pyrexia, or the case is complicated with phthisis or regurgitant heart disease, the urine may be high-colored, and turbid from lithates.

The quantity of urine voided per day varies according to the type of the disease, and the presence or absence of pyrexia, sweating, vomiting, or diarrhœa. The urine is throughout scanty with the large white kidney; I have known it not to exceed 35 ounces on any one day for a period of four months, and to be under 20 ounces for three successive weeks, and under 12 ounces for several consecutive days. It may even sink to one or two ounces. With the granular contracting kidney, the urine is abundant (three or four pints a day) in the middle periods of the disease; but it gradually grows scantier towards the termination; in exceptional instances, the diuresis is profuse, and the urine may occasionally amount to five or even nine pints a day.[1] These larger quantities have been generally observed after an attack of uræmic convulsions, or coincidently with sudden subsidence of dropsy.

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[1] Hamilton, out of sixty cases of scarlatinal dropsy observed by him in Edinburgh, encountered two, in which there was no albuminuria.

The specific gravity is low when the urine is copious (1006 to 1015); but when it is scanty, the sp. gr. may rise to 1030 or even 1040.

The reaction of the urine is nearly always acid; and, not unfrequently, it deposits uric acid and oxalate of lime. Occasionally I have noted it alkaline from fixed alkali, and twice ammoniacal on emission.

The *renal derivatives* (epithelium and tube-casts) are markedly scantier in the chronic, than in the acute forms of Bright's disease; and it is not uncommon for them to be altogether absent for limited periods. They are, however, sometimes discoverable when the urine has ceased (temporarily) to be albuminous. The appearances of the discharged epithelia and casts present considerable diversities, which supply an important insight into the structural changes going on within the kidney. The epithelial cells may be simply withered; more rarely they are totally disintegrated into an amorphous granular débris; in other cases they contain specks of oil, or they may even be wholly converted into an agglomeration of oily particles so as to appear identical with the so-called "granular" or "Gluge's corpuscle." The casts are sometimes similarly speckled with fat, and free oily dots are scattered over the field. Such a conjunction indicates a fatal disorganization of the organs—either large fat kidneys, or contracted granular ones. But the casts *most commonly* seen in chronic Bright's disease are "small" and "large" hyaline forms, and "granular" opaque ones. Any of these may have a few wasted epithelial cells strewed over them. Perfect "epithelial" casts are rare in chronic cases, and blood casts are still more rare, unless there be concomitant tricuspid regurgitation.

When intercurrent exacerbations of the renal process, with pyrexia, arise, there will be found (whatever may have been the previous character of the casts) medium-sized and large solid-looking, pale-straw, albuminous casts resembling molten glass (*see* Fig. 55).

Casts of these diverse appearances may be discharged by the same individual, even during the same day. Conclusions as to the probable state of the kidney can only be drawn from the *prevailing* character of the deposit, and not from one or two individual casts or cells. This diversity in the character of the casts arises from the different condition of the several parts of the gland. In some portions the tubuli may be denuded of their epithelium, and the exudation thrown into them is discharged in the form of large hyaline casts; if the denuded portions have undergone subsequent contraction the casts will be small and hyaline. Other tubes, clothed or partly clothed with epithelium, shed some of their cells with the contained exudation, and cause the appearance in the urine of casts more or less studded with epithelial remnants. The longer the exudation is retained within the tubuli, the darker and more granular will it appear, when discharged as casts; and *vice versâ*, casts speedily discharged are commonly hyaline. Sometimes casts are darkened by the coloring matter of the blood; and the opaque granular ones are (sometimes at least) composed of crushed epithelial débris moulded into the form of the tubuli. See Diagnosis.

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[1] Christison, pp. 174 and 186. Pfeufer, in Henle and Pfeufer's Zeitsch., Bd. I. p. 58.

The normal solids of the urine are all diminished in chronic Bright's disease. The urea, is, as a rule, markedly reduced—the daily quantity averaging only about 100 grains; Frerichs has observed it as low as 15 grains.[1] There is no correspondence, direct or inverse, between the excretion of urea and the discharge of albumen. With intercurrent pyrexia the excretion of urea rises.

The changes in the *blood* are the complement of those in the urine. The blood becomes more watery and poorer in albumen and red corpuscles. On the other hand, urea, uric acid, the extractive matters and the pale corpuscles accumulate in it. This alteration in the composition of the blood is deeply concerned in the production of the more prominent features of the disease—the anæmia, dropsical effusions, uræmic phenomena, and secondary inflammations.

Dropsy is much oftener absent in the chronic than in the acute form. It is much more constant with the smooth large, than with the granular contracted kidney. Of the latter class probably one-third or one-fourth of the cases run their entire course without dropsy. The effusion begins quite as often in the feet and legs as in the face; it is commonly slight and partial, but sometimes excessive and general. When the heart or liver is diseased, ascites and œdema of the legs become disproportionately prominent. The effusion is apt to change its seat capriciously; and it comes and goes from time to time. Sometimes it disappears totally for months, and then returns again. More frequently, after a subsidence of the general dropsy, œdema lingers obstinately in one or two places—over the flat of the tibiæ, about the ankles, beneath the eyelids, under the conjunctival membrane, or about the genitals. The presence or absence of dropsy, generally, but by no means always, corresponds with the abundance or scantiness of the urine; but it has no relation to the amount of albumen.

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[1] Exceptions occur to this rule. Mosler mentions a case of Bright's disease in which 640 grains of urea were voided in one day (Archiv d. Vereins, Bd. xi. S. 513). Schottin found creatine and creatinine increased in the urine in Bright's disease, and the increase was observed to keep pace with the intensity of the uræmic symptoms (Archiv der Heilk., 1860, p. 417).

The *skin* is usually obstinately dry, perspiration is quite exceptional; and when it occurs, is commonly due to diaphoretic measures of treatment. Profuse sweating does, however, sometimes take place spontaneously, and may even continue for weeks. In one such case under my care an abundant crop of pemphigus vesicles broke out on the surface. The integuments in some cases are excessively pale and glossy, but more commonly they are sallow and rough. There is little or no tenderness in the renal region in the chronic cases, and the frequency of micturition is mostly observed at night.

Some degree of *bronchitis* is almost an invariable coincident of Bright's disease both in the acute and chronic form.

The *pulse* in Bright's disease almost invariably reveals high tension of the arterial system, when examined either by the finger or by the sphygmograph. The high tension is shown not by the pulse alone, but also by certain auscultatory signs, amongst which are accentuation of the aortic second sound, and, as was pointed out by the late Dr. Sibson,[1] a reduplication of the first sound of the heart, at the cardiac apex. Dr. Broadbent and Dr. Mahomed have shown that such high tension may precede all other signs of Bright's disease, and appropriate treatment may indeed ward off the more serious manifestations.

The *retina* in Bright's disease, and more frequently in cases of the red granular kidney, shows various changes, some of which are characteristic. Dr. Gowers[2] first described a narrowing of the retinal arteries, coincident with the high blood pressure, the narrowed arteries frequently showing a white border. Inflammation of the optic nerve, and diffused inflammation of the retina are met with; hemorrhages, too, are frequently seen and are most frequently "flame-shaped," and situated in the nerve fibre layer. The most characteristic lesion, however, is a peculiar appearance of white specks arranged in radiations from the yellow spot. These specks are due to degeneration of the nerve fibres. The renal affection always precedes the retinal changes, although the latter have in many cases first called the attention of the observer to the possibility of Bright's disease being present.[1]

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[1] Brit. Med. Journ., 1877, I. p. 33.

[2] Ibid., 1876, II. p. 743.

COMPLICATIONS AND CONNECTION WITH OTHER DISEASES.—In long-standing cases hemorrhage from the various mucous surfaces sometimes occurs. A little hæmoptysis is not infrequent, and occasionally severe epistaxis. The digestive organs are nearly always disturbed: at first there is loss of appetite and nausea; in the later periods frequent or even uncontrollable vomiting is not uncommon. The bowels are alternately bound and loose. Severe fitful diarrhœa, which leaves the dropsy undiminished, is not uncommon, especially towards the close of the complaint. Not unfrequently, anatomical lesions are found in the *intestines* which explain these disturbances; in other cases they are manifestly uræmic. Treitz states that urea is discharged into the intestines from the blood, and converted into carbonate of ammonia, which acts as an irritant on the intestinal mucous membrane. The more palpable changes found in the intestines are, follicular catarrh, dysenteric ulcers—sometimes with sloughing of the mucous membrane. In 220 cases of Bright's disease collected by Treitz, the following conditions of the intestines were found after death.[2]

Hydrorrhœa (intestines filled with yellow-greenish fluid) .	80	times.
Blennorrhœa and catarrh	60	"
Croupous and ulcerous dysentery	19	"
Sloughing	12	"
Sanguineous contents without discoverable source of hemorrhage	4	"
Normal feces	5	"
Contents of intestines undetermined.	11	"

Secondary inflammation of the lungs, endocardium, pericardium, pleura, peritoneum, or integuments, may break out at any period in the course of chronic Bright's disease. The tendency to these constitutes one of the principal dangers of the complaint. Cardiac hypertrophy, valvular disease, and pulmonary tubercle are frequent complications.

The following table exhibits the proportionate frequency with which the various organs, other than the kidneys, are found affected in Bright's disease generally. It contains the result of 406 autopsies, contributed as follows: Bright, 100; Christison, 14; Gregory, 37; Martin-Solon, 8; Rayer, 48; Becquerel, 45; Bright and Barlow, 10; Malmsten, 9; Frerichs, 21; Rosenstein, 114.

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[1] *See* J. Clifford Allbutt—On the use of the Ophthalmoscope, and Gowers's Medical Ophthalmoscopy.

[2] Prag. Vierteljahrschr., 1859.

Heart.	Lungs.	Pleura.	Pericardium.	Peritoneum.	Liver.	Spleen.	Stomach and Intestines.	Brain.
125 times hypertrophy. 54 times with valvular disease. 55 times without valvular disease.	115 times œdema of lungs. 52 pneumonia. 8 pulmonary apoplexy. 4 gangrene 37 tubercle. 33 vesicular emphysema.	57 pleurisy.	30 pericarditis.	46 peritonitis.	41 cirrhosis. 38 fatty liver.	58 chronic tumor. 17 acute splenic tumor.	36 gastric catarrh. 85 catarrh and follicular ulceration of intestine 13 tuberculosis of intestine	14 sanguineous apoplexy. 59 effusion of serum under arachnoid-

In addition there were: 1 case complicated with cancer of the liver; 4 cases with cancer of the pylorus; 2 typhoid ulcers of intestines; 2 meningitis; 1 meningeal tubercle; 11 tumor cerebri; 3 abscess of lung; 11 nutmeg liver; 3 lardaceous liver; 9 contraction of spleen; 3 diphtheritis of intestines; 1 softening of brain; 6 chronic arachnitis; 1 suppurative meningitis.

Bright's disease and phthisis. This complication is of frequent occurrence. In the great majority of cases the pulmonary disease is advanced to its later stages before the renal symptoms make their appearance. The long-continued discharge of pus from the lungs gives rise at length to waxy changes in the kidneys, which are followed by albuminuria and dropsical effusion. This is no doubt the usual history of such cases; but sometimes the renal disease precedes the pulmonary, and the changes found in the kidneys after death are not invariably of the waxy type.

The coexistence of two fatally tending diseases might have been expected to accelerate the inevitable issue; yet most cases of this class have an exceedingly chronic course, and continue in a stagnant condition for months together. The onset of albuminuria often determines a fall of the temperature.[1] In the following remarkable instance, the pulmonary disease (already in its third stage) completely retrograded, and was supplanted by the renal affection.

M. C., æt. 20, a mechanic, was admitted into the Royal Infirmary, October 27, 1863. He was a well-grown young man, with white pallid features, dry skin, heavy eyes, and moderate œdema of the lower extremities. The abdomen was enlarged from ascites, and the integuments of the flanks and hypogastrium were œdematous; pulse 112, regular, small; respirations 21; tongue moist, slightly furred. The state of the chest on admission was as follows: Diminished expansion over both apices; but more on the right side than the left; conspicuous depression of the right infra-clavicular region. There was almost complete loss of resonance on the right side as low as the second interspace. The right upper scapular regions were also dull on percussion. Moist crepitation and cavernous rhonchi were heard beneath the clavicle on both sides. Whispering pectoriloquy was very distinct below the right clavicle, and present, though less typically, over the left apex. The heart's sounds were natural; there was no appreciable hypertrophy. The expectoration was copious, airless, purulent.

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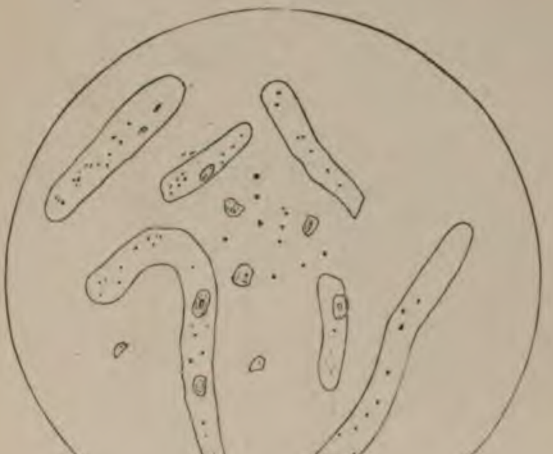
[1] *See* Williams, Brit. Med. Journ., 1883, II. p. 1224.

The urine was scanty, amber-colored, specific gravity 1030, intensely albuminous, becoming almost solid on boiling. A slight deposit of withered renal epithelia and transparent tube-casts, without any, or only very faint, signs of fatty changes, lay at the bottom of the glass.

The history disclosed perfect health until ten months ago, when the patient began to cough. He attributed these symptoms to cold taken by passing out into the cold air from his hot work-room. His family is tuberculous; a sister came subsequently under my care with phthisis. He had night perspirations six months ago. Three weeks before admission the ankles began to swell; but the skin had been dry for three months.

Cod-liver oil and iron were prescribed; a warm bath was administered every other evening. The patient constantly kept his bed, on account of the swelling in his legs increasing when he sat up.

FIG. 54.



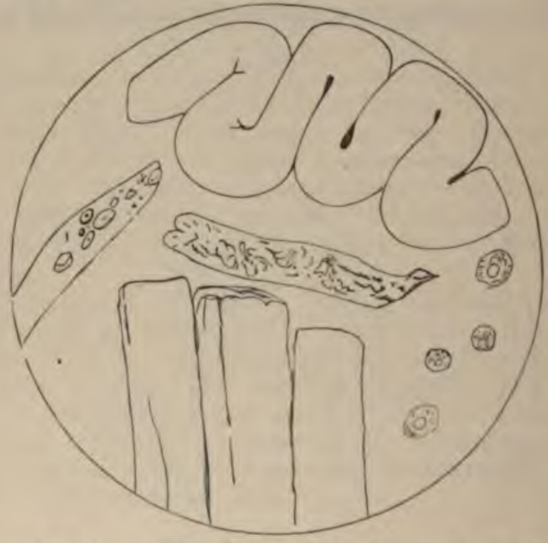
Transparent hyaline casts, from the urine of M. C., on January 29 (quiescent period).

For a period of two months I was unable to attend at the Infirmary on account of illness, but the treatment was carried on during my absence without alteration, and the patient kept continuously in bed. When I revisited the wards in January, 1865, I found the renal symptoms somewhat advanced; but the pulmonary complaint had decidedly receded. The urine was very scanty, varying from 12 to 18 and 26 ounces a day, with a specific gravity ranging from 1030 to 1034; it often deposited amorphous urates. On January 29th the urinary deposit corresponded to the following description: It was scanty, and composed of atrophied renal cells, with a few excessively transparent small hyaline casts, some of which were speckled with albuminous granules and a few doubtful oil particles (*see* Fig. 54). The patient at this date was in a quiescent state and free from fever.

The chest complaint was now altogether in the background; there was scarcely any expectoration, and the physical signs indicated a marked amelioration. The depression under the right clavicle was less conspicuous, and the movement improved; the percussion sounds were still unaltered, and the rhonchi still cavernous, but not abundant; pulse varied from 88 to 100; respiration from 20 to 22.

During February the urine became still scantier (12 to 20 ounces a day), with a density ranging from 1033 to 1041. It became almost solid on boiling. The anasarca increased, and extended into the face and upper limbs. Occasional vomiting took place, and the appetite failed entirely.

FIG. 55.



Massive molten-looking casts, from the urine of M. C., February 28 (pyrexial period).

In the last week of February the patient insisted on going home. But he had not been out a single day before he took a violent cold, ushered in with repeated shiverings. The anasarca increased rapidly; respiration became oppressed, and he was readmitted three days later (February 26th) in the following state: Great general dropsy, the urine almost suppressed, distressing oppression of breathing. A compound jalap powder was administered; after which he vomited and had three loose motions.

On the following day, the patient was very thirsty and feverish; tongue furred, red at edges; pulse 128; respiration 30; cough very distressing; a scanty expectoration of nummular purulent sputa. He complained loudly of pains in the abdomen, chest, and back, especially when he turned in bed. There was great restlessness.

A hot-air bath was administered with the effect of inducing copious sweating, and reducing the œdema somewhat.

February 28.—Urine, last 24 hours, only 8 ounces; it was intensely albuminous and deposited urates. The renal derivatives presented totally new characters; they are delineated in Fig. 55; the new feature was the appearance of massive molten-looking casts of large and medium size. Some of them were slightly granular in spots; a few were also sparsely studded with epithelium; but there were no proper "epithelial casts;" there was neither blood nor fat. Some of the large casts lay side by side like thick logs, and appeared as if split in a longitudinal direction at their extremities (Fig. 55).

On the 28th the patient grew feebler and more restless; obstinate vomiting set in, and continued nearly till death, which took place on the morning of the 29th. Only two ounces of urine were passed in the last 24 hours of life. There were neither convulsions nor coma; and vision continued good to the last.

Autopsy, 30 hours after death. The *right lung* presented an exquisite example of retrograde phthisis. Half a dozen small cavities were counted in the upper lobe—all of them small, varying from the size of a pea to that of a horse-bean—completely lined with a thick pyogenic membrane. Not a particle of tubercle existed around these cavities nor in any part of this lobe. The pulmonary tissue was dark and leathery, and very imperfectly aërated. The right apex was condensed, deeply puckered, and traversed in various directions by thick white lines of cicatricial tissue. In the lower lobe of the same lung, a vomica as large as a filbert was found with anfractuous boundaries composed of tuberculous matter. Small masses of obsolete tubercle—some cretaceous, others putty-like—were scattered sparsely through the lower lobe.

The *left lung* was crepitant throughout. The upper lobe contained three cavities—one as big as a walnut—lined with pyogenic membrane, and not surrounded by tubercle. Small nodules of tubercle were scattered through the upper and middle lobes—some cretaceous, some putty-like, others unsoftened and crude. The inferior lobe of the left lung was highly œdematous. No fluid existed in either pleura; but old adhesions prevailed sparingly on both sides.

The *heart* was of the usual dimensions; the walls of the left ventricle were thicker than was to be expected in a case of phthisis. Both sides were filled with firm bulky clots of yellowish fibrine, which closely adhered to the inequalities of the chamber, and sent voluminous prolongations into the aorta and pulmonary artery. The formation and presence of these clots evidently constituted the immediate cause of dissolution.

The *liver* was large and pale; the hepatic cells well formed, and not containing more than the usual quantity of fat molecules.

The *spleen* was larger than usual; its texture soft and natural.

The *kidneys* weighed together 23 ounces, and the two were almost exactly of a size. They furnished a typical example of the large, smooth, mottled kidney. Their surface was perfectly smooth; the capsule, thin and transparent, peeled off readily without tearing the glandular tissue. The prevailing color of the surface was fawn, marbled here and there with red; the fawn color was picked with dead white, as in ivory. The organs were conspicuously soft and flabby. The red parts of the surface showed minute spotty and sinuous injection of the superficial vessels.

On section, the cortex was found greatly hypertrophied; it stood half or three-quarters of an inch thick on the broad ends of the cones. It had a full fawn color, with broken streaks of red running through it in diverging lines, from the bases of the pyramids. The pyramids were unusually pale, though from the exsanguine state of the cortex, they offered a pretty strong contrast of color with the latter. The epithelial lining of the convoluted tubes was extensively disorganized; both cell and nucleus were reduced to a granular, fatty débris. Scarcely a single cell approaching perfection could be seen. The Malpighian corpuscles were not altered in size; but they were penetrated, and rendered opaque, by a granular material. The epithelium of the straight tubes was in much better preservation; not only the nuclei could be seen, but the outlines of the cells themselves. A considerable quantity of spindle-shaped fibre-cells were found, also medium-sized, massive-looking casts—resembling those found in the urine shortly before death.

Two things appeared singular in relation to these kidneys, namely, that the urine should contain so few renal derivatives when the kidney contained such an immense quantity in a disorganized state; and secondly, that this disorganized material should contain fat in such quantity without there having been any, or scarcely any, in the urinary deposit. Perhaps the degeneration of the epithelium only attained this maximum degree in that last intercurrent febrile attack which immediately preceded death; if so, the plugging up of the canals of the pyramids with the massive casts, which then appeared for the first time in the urine, and which may have been the determining condition of the suppression of urine, would cause the absence from it of the derivatives of the convoluted tubes.

BRIGHT'S DISEASE AND DISEASES OF THE HEART AND VASCULAR SYSTEM.—The connection of cardiac disease with renal disorder is at least threefold.

In the *first* class of cases, simple *hypertrophy* of the heart and especially of the left ventricle, is found without valvular incompetency and without degeneration of the muscular fibres. In this class, which is a numerous one, as the table at p. 420 shows, the cardiac affection is secondary to the renal.

Bright, who was the first to point out this curious coincidence,[1] offered two explanations of it—either, that the altered composition of the blood exercised an irregular and unwonted stimulation upon the muscular tissue of the heart, or so impeded the circulation in the capillaries, that a greater effort of the ventricle was required to drive the blood through the distant minute branches of the bloodvessels. Traube[1] explains the occurrence of simple cardiac hypertrophy in chronic Bright's disease somewhat differently. In his experience the kidneys in these cases are markedly atrophied.[2] The contraction of the renal tissue involves destruction of a certain amount of secreting structure and a diminution of the flow of blood through the organs by reason of the destruction of bloodvessels. Two consequences follow, namely, that a diminished amount of blood passes from the arterial into the venous system, and that a less quantity of fluid is withdrawn from the arterial system for the formation of urine. Both circumstances, but especially the second, operate to increase the tension in the arterial system, and consequently to increase the resistance which the left ventricle has to overcome in discharging its contents. The hypertrophy which follows is, therefore, according to Traube, a conservative or compensating change, similar, in the mechanism of its production, to that induced by valvular incompetency or aortic constriction. If the compensation be complete, the heightened tension in the arterial system occasions a larger transudation of water and even of urea and other urinary solids, through the kidneys, and in that manner materially helps to stave off dropsical effusion and uræmic symptoms. But should some additional obstruction to the circulation arise, through intercurrent inflammation of the bronchial tubes, or of the lungs, pleura, or pericardium, the heart—enlarged and strengthened through it be—no longer suffices to overcome the increased resistance, and dropsical effusions or uræmia speedily make their appearance. Traube adduces some apposite examples in which individuals with contracted kidneys enjoyed fair health, with capability of exertion, and continued free from anasarca and uræmic disturbance, until the advent of some complication disordered the balance of the circulation, and *then* the urine became scanty, and the familiar symptoms of renal disease, previously latent, broke forth into prominence.

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[1] Guy's Hospital Reports, vol. i. p. 396.

The first part of Traube's argument, that the granular kidney offers a resistance to the passage of arterial blood, has been proved by the experiments of Dickinson and Thoma mentioned above (*see* page 400); but whether this obstruction is competent to produce high arterial tension and hypertrophy of the heart is doubtful. Experimental evidence is conflicting. Ludwig ligatured the renal arteries without causing high vascular tension or hypertrophy; Grawitz and Israel narrowed the renal artery and removed one kidney, but while they did so cause hypertrophy of the heart, they assert that there was no increased tension. Lewinski,[3] on the other hand, by similar experiments obtained both hypertrophy and increased tension. Bamberger objects to the first part of Traube's view, on the grounds that hypertrophy of the heart may be found when there is no destruction of the bloodvessels in the kidney, or where anastomoses have been set up, as was shown by Thoma in the granular kidney; that the ligature of even large vascular trunks does not cause hypertrophy; and that this view does not apply to the hypertrophy of the right ventricle, which is also found. Bamberger[1] adopts the second part of Traube's view, that the hypertrophy is produced by an increased volume of the blood caused by deficient excretion of fluids. In the granular kidney, where the urine is increased in quantity, he believes that a stage of deficient excretion has preceded, and that the present hyperexcretion is merely due to the balance of the system being set for a higher grade.

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[1] Ueber den Zusammenhang von Herz- und Nieren-Krankheiten, p. 58.

[2] In a later communication Traube brings forward evidence to show that hypertrophy of the left ventricle is an almost constant concomitant of granular and contracted kidney. In 77 cases collected by him from various sources, the left ventricle was found hypertrophied in 93 per cent. (Deutsche Klinik, 1859, p. 315). In 187 cases of granular kidneys collected by Dr. Sibson enlargement of the left ventricle (without valvular disease) was found in 128.

[3] Zeitschr. f. klin. Medic., vol. i. p. 561.

Dr. George Johnson has discovered that there is a wide-spread hypertrophy of the muscular walls of the small arteries in chronic Bright's disease. He finds it not only in the arteries of the kidneys, but also in those of the pia mater, the skin, the intestines, and the muscles. He thus explains the production of this hypertrophy and the manner in which it reacts on the left ventricle: "In consequence of the degeneration of the kidneys the blood is morbidly changed. It contains urinary excreta, and it is deficient in some of its own normal constituents. It is therefore more or less unsuited to nourish the tissues—more or less noxious to them. The minute arteries throughout the body resist the passage of this abnormal blood. The result of this antagonism of forces is, that the muscular walls of the arteries and those of the ventricle of the heart become simultaneously and in an equal degree hypertrophied. The persistent overaction of the muscular tissue, both cardiac and arterial, is found registered after death in a conspicuous and unmistakable hypertrophy."

With this view Dr. Broadbent in the main agrees. He believes, however, that the vitiation of the blood precedes the kidney change. During this prenephritic stage, high tension is produced by the contraction of the muscular walls of the arterioles, but by appropriate treatment the kidney mischief may be avoided. Dr. Broadbent shows that the high tension, even in confirmed Bright's disease, is not permanent, but may temporarily disappear during an attack of pyrexia, or under the influence of nitrite of amyl, which relaxes the arterioles. This tends to show that it is the action of the arterial muscular tissue which produces the high tension, and not a permanent change, such as the capillary fibrosis of Gull and Sutton, mentioned below.

Dr. Galabin believes that the impediment to the circulation in chronic Bright's disease lies not as Dr. Johnson supposes in the small arteries, but in the capillaries, and is due to a modification of the capillary attraction between the blood and the walls of the vessels, and that, the arterial pressure being thus increased, the muscular walls of the heart and arteries are both hypertrophied in concert, since both have to act against greater resistance.

Sir Wm. Gull and Dr. Sutton have advanced a novel view of the pathology of granular kidney and of the associated changes in the cardio-vascular system. They announce the discovery of a new pathological change, to which they give the name of "arterio-capillary fibrosis." This consists in the deposit of a "hyalin-fibroid" material in the fibrous coats of the arterioles and capillaries. This change may prevail extensively throughout the vascular system—in the kidneys, skin, pia mater, heart, lungs, spleen, stomach, and retina. In its nature it is allied to but not identical with senile changes. It commonly begins in the kidneys, but it may begin elsewhere; so that the hypertrophy of the heart with degeneration of the bloodvessels may be found associated with healthy kidneys; and that when atrophy with granulation of the kidneys exists it is but part and parcel of a general morbid change. The granular form of Bright's disease is, in their eyes, essentially an example of arterio-capillary fibrosis. This view has been strongly supported by Dr. Mahomed in this country, and by Leyden in Germany.

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[1] Volkmann's Samml. klin. Vorträge, No. 173.

That a wide-spread change in the small arteries of the body exists in Bright's disease seems now well established, but what the nature of that change may be is by no means determined. Thoma showed that there was thickening of the subendothelial layer of the small arterioles, corresponding to the similar lesion found in syphilis by Huebner. This condition of the vessels, so far at least as the kidney is concerned, is generally admitted. Thoma was of opinion that the muscular coat instead of being hypertrophied was really atrophied. Ewald, who examined the vessels of the pia mater covering the pons Varolii in sixty-two cases of Bright's disease, could not confirm the discovery of Gull and Sutton. The appearances described by them, he believed, were the results of the reagents employed. On the other hand, he confirmed the opinion of Dr. G. Johnson as to the hypertrophy of the muscular coat, and showed further, that the general vascular change was associated rather with interstitial than with parenchymatous changes in the kidney.

Senator described a difference in the character of the hypertrophy according as the kidney change began in the epithelium or in the interstitial tissue. In the former case dilatation was combined with hypertrophy of the heart, while in the latter the hypertrophy was pure. In the first case he believed with Dr. G. Johnson that the retention of excrementitious matters in the blood caused a rise of arterial tension and consequent hypertrophy; in the second case he believed the hypertrophy to be due to a general change in the small arteries. His differentiation of the two kinds of cardiac enlargement cannot, however, be sustained, for dilatation of the left ventricle is by no means uncommon in the later stages of granular kidney, and is due to weakening of the heart muscle.

Buhl[1] placed the cardiac change in the primary position. He believed that there was initially a myocarditis which caused the hypertrophy. Debove[2] and Letulle, in support of this view, have shown that a fibrous growth between the muscular fibres of the left ventricle is common in Bright's disease. The opinion has not met with any considerable support.

These views have been sharply criticised, and are still in dispute, but they unquestionably bring into true prominence what every observer of extended experience in cases of granular kidneys must have had strongly impressed on his mind—namely, that the renal disease is not merely a local affair, but that it is, rather, a part of a widespread tissue degeneration involving the entire, or a large, portion of the body.

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[1] Mittheilungen a. d. path. Institut, München, 1878.

[2] Progrès Médical, 1876, No. 52.

Of the various forms of chronic Bright's disease, the red granular kidney is the most frequently accompanied by hypertrophy of the heart; less frequently does the association occur with the large white kidney; while with the amyloid kidney the enlarged left ventricle is only rarely found.

In the *second* class of cases, *valvular* defects and their consequences coexist with Bright's disease. Most of these are examples of endocarditis, secondary to the renal disease. But in other cases the cardiac and renal affections arise independently of each other, and depend on some cause common to both—as in the following example:

J. H., æt. 48, was admitted into the Manchester Royal Infirmary, November 22, 1862. He had right hemiplegia; the mental faculties were wholly disordered; there was gay incoherence and insanity; no fever. The heart's apex beat in the fifth interspace, half an inch outside the nipple line; a loud systolic bruit was audible at the apex and the mid-sternal base, and extended up the aorta; a faint diastolic bruit was audible over the second right cartilage. The urine was albuminous to about one-fifth; there was no dropsy.

The patient remained in the Infirmary a month. The mental derangement subsided in ten days, and perfect coherence returned. The other symptoms remained unchanged. He returned home; and in a few days was seized with coma, which proved rapidly fatal. The autopsy revealed: granular red kidneys with abundant presence of fat; cardiac hypertrophy with fatty degeneration of the muscular fibres; extensive disease of the mitral and aortic valves with atheromatous patches on the aorta; two old apoplectic clots were found in the left hemisphere, and wide-spread fatty degeneration of the arteries existed at the base of the brain.

In this instance fatty degeneration had simultaneously invaded the heart, the brain, and the kidneys, and produced a triple series of symptoms—all essentially independent of each other.

The *third* class of cases are those in which the renal disorder (congestion, etc.) is secondary and subordinate to cardiac disease. These cases have already been fully described in Chap. I. in connection with Congestion of the Kidneys, which see.

URÆMIA.

Certain phenomena, chiefly affecting the nervo-muscular system, arising in the course of Bright's disease, have been attributed to a poisoned state of the blood, from the retention in it of excrementitious matters which the disabled kidneys are unable properly to eliminate. To these phenomena the term *uræmic* has been applied; they consist of twitchings and convulsions of the voluntary muscles, headache, drowsiness, coma, defects of sight and hearing, vomiting and diarrhœa.

It is a marked feature of uræmic phenomena that those which are of a paralytic nature affect the sensorium and the special senses, but not the voluntary muscles; while those of an opposite kind (exalted irritability) affect the voluntary muscles, but not the sensorium. Delirium is rare, while coma is frequent; paralysis of the limbs is scarcely known (unless there be some anatomical lesion of the brain superadded), while convulsions are frequent.

The mode in which uræmic symptoms enter on the scene, and the forms they assume, present great diversity.

Generally they begin insidiously with headache or vomiting, followed by heaviness, indifference, and somnolence. These premonitories may either pass away in a few days without further consequence, or they may be succeeded by general convulsions and coma. In other instances the patient is at once struck down with convulsions or insensibility without any previous warning, or he becomes suddenly blind, or is seized with uncontrollable vomiting.

The most common of these symptoms is *headache;* few individuals with degenerated kidneys altogether escape it. A sense of heavy weight or compression is complained of over the forehead or vertex. Sometimes the pain is obstinately fixed at the back of the neck, or behind the orbits.

The *defects of sight* consist either in a dimness of vision (amblyopia), which comes and goes—objects appearing as if veiled in mist—or in rapid and complete, though usually temporary, blindness. The convulsive seizures are often accompanied with temporary loss of sight, which generally persists in greater or less degree for a certain time after the spasms have passed away.

The ophthalmoscope reveals no organic change in the eye in genuinely uræmic amblyopia: it is a purely cerebral phenomenon, and not to be confounded with the hemorrhagic blindness (Retinitis apoplectica), which is also not uncommon in Bright's disease. and which is due, as V. Graefe has shown, to rupture of the retinal vessels. In this latter affection (which is in no sense uræmic) the loss of sight is seldom complete, but is of a more permanent character. The production of it is probably due to hypertrophy of the left ventricle which so commonly accompanies a contracting kidney, and the increased tension in the arterial system consequent thereupon; it is an occurrence of the same order as the sanguineous apoplexy to which the same individuals are liable.

Uræmic *deafness* is much less common than amblyopia, and its occurrence is highly exceptional.

Uræmic *convulsions* are of the epileptic type, and, as a rule, they conform strictly to that type—being accompanied with complete insensibility, rolling of the eyes, biting of the tongue, and foaming at the mouth. The paroxysms commonly leave the patient deeply comatose.

In exceptional instances consciousness is not wholly lost. In a lady under my care the paroxysms coincided with the catamenial periods; during the convulsions the patient knew the persons about her, and called loudly to be held fast. A case is related by Bright in which the spasms at first resembled cramps; these were followed by twitchings of the hands, arms, shoulders, chest, and legs. The spasms were almost constant, and caused a somewhat hurried mode of expression when the patient spoke, but the intelligence was perfect. As the case proceeded the spasms became more and more severe, with forcible drawing up of the legs, and distortion of the muscles of the face; the faculties were retained to the last.[1]

An attack of uræmic convulsions may consist of only a single paroxysm: more frequently there occurs a succession of paroxysms or fits, following each other at uncertain intervals of a few minutes or several hours—the patient lying during the remissions in a state of profound insensibility, with stertorous breathing, pale face, and dilated pupils; or in deep drowsiness, but capable of being partially roused, when spoken to or shaken.

If a first attack does not prove fatal, it may recur at irregular intervals of weeks or months, or be replaced by uræmic symptoms of some other order. Instead of the clonic convulsions tonic spasms are occasionally met with, affecting various muscles. Occasionally, too, the spasms are limited to special groups of muscles and may occasionally cause a condition of opisthotonus.[2]

Uræmic *coma* either creeps on very gradually, passing on, in the course of two or three days, into complete stupor; or it culminates quickly—the patient falling down, as if in apoplexy, perhaps while walking in the street, or occupied with his usual avocation. Cases of this class when there is no anasarca, and the previous state of the urine is unknown, are very liable to be confounded with apoplexy or with narcotic poisoning. The following instructive illustrations of such an occurrence are related by Mr. Moore and Dr. Richardson:

CASE 1.—An old soldier, named Price, was received into Queen's Hospital, Birmingham, with the following symptoms: breathing laborious and sometimes stertorous; when left alone the patient passes into a state of stupor, answers questions sensibly when roused; pupils moderately dilated, indolently sensitive to light.

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[1] Guy's Hospital Reports, 1840, p. 139.

[2] Jaccoud. Pathol. Interne, 6th edit., vol, ii. p. 439.

It appeared that Price, having suffered some days from diarrhœa, went into a druggist's shop, and asked for a pennyworth of tincture of rhubarb. The shopman added to this dose a few drops from another bottle, and Price swallowed the whole before leaving the shop.

Immediately after taking the above dose he became drowsy and vomited; at the suggestion of a neighbor he returned to the shop, and asked the shopman whether he had given him laudanum. The latter told him that he had put in a few drops on account of the severity of his symptoms. Upon again reaching home he fell asleep, and continued sleeping unless temporarily roused. In this state he was taken into hospital, and was treated as a case of opium poisoning. A mustard emetic was ordered immediately; the patient was kept in constant motion, and plied with strong coffee. He improved considerably under the treatment, and talked over his old campaigns with the porter who had charge of him. Next day he relapsed into a lethargic state; galvanism was employed without benefit; he was now walked round the hospital garden between two men, and strong infusion of green tea was administered. A little improvement followed, but at 2.30 P. M. he relapsed once more, and the breathing became more oppressed. As long as he was kept moving he could be made to answer questions; but in the course of the afternoon the somnolence deepened in spite of the treatment. Ether and ammonia were applied to the nostrils; cold water was dashed over the face and neck; but at 8 P. M. the drowsiness had become insuperable; the stertor augmented. Mustard poultices were put to the legs. Venesection was tried, but when four ounces of blood had flowed the pulse became thread-like, and it was thought prudent to desist. The coma increased in intensity; and he died at 2.30 A. M., 44 hours after admission, and 102 hours from the time of taking the dose.

A coroner's inquest was held on the case, on account of the suspicion of poisoning; but the results of the post mortem went to exonerate the druggist, for the kidneys were found granular and greatly atrophied, and the urine left in the bladder was found albuminous. (J. Moore, "London Med. Gaz.," 1845, p. 821.)

CASE 2.—A woman, aged 34 years, who was given to drinking, and had recently been treated for primary syphilis, was seized on November 18, 1859, with rigors. She was attended by a neighboring chemist, who, on November 22d, gave her a mixture which, he said, contained dilute nitric acid, nitrate of potassa, syrup of buckthorn, sulphate of magnesia, and water. It was afterwards proved by analysis that these were the constituent parts of the remaining portion of the mixture. The medicine was sent in to the woman on the evening of the 22d, and after taking a large quantity of beef-tea, she swallowed one dose of mixture. Five minutes afterwards she became hysterical and convulsed, and the friends believing the woman to have been poisoned, summoned the druggist, who in alarm tried to get her to take some ipecacuanha, but without avail. In the course of the night a medical man was called in; he found the woman in a state of typhoid coma, with pupils slightly dilated and immovable, and the body at times convulsed—the convulsions assuming an epileptiform type. Every available means of treatment was carried out, but the coma became more profound, and seventy hours after the administration of the mixture above mentioned, death closed the scene. By the coroner's warrant the body was examined, and a chemical inquiry instituted. The brain was quite healthy; the kidneys were greatly diseased—large, flabby, pale, speckled, soft, and greasy. The analysis disclosed no poison of any sort. (Richardson, "Clinical Essays," p. 135.)

CASE 3.—A gentleman, æt. 63, was driving in an open chaise through the village of Mortlake, in 1853; he was observed by his servant, who was by his side, to be constantly drowsy; at last he suddenly seemed to fall into a helpless state and dropped from the chaise. He was conveyed into a house, and Dr. Richardson was summoned. Dr. R. found him suffering from all the signs of narcotic poisoning; the pupils were fixed and slightly dilated. Some urine was withdrawn from the bladder, and found to be largely charged with albumen. He recovered from the attack; but three weeks later he suffered again in the same way, and died with typhoid coma—the urine being altogether suppressed for many hours before death, and having been albuminous throughout the illness. (Richardson, "Clinical Essays," p. 141.)

The *diagnosis* of uræmic coma from apoplexy rests on the absence (in the former) of paralysis, and the partial recovery of consciousness between the convulsive attacks—if there be any. From poisoning by opium, renal coma is distinguished by the dilated or semi-dilated state of the pupils, and by the occurrence of remissions in the insensibility. From ordinary epilepsy the diagnosis—apart from the antecedent history, which, if known, suffices to indicate the nature of the case—is sometimes difficult. The incidents of the seizures are often identical, even to the existence of an aura. In uræmia the respiration frequently assumes the Cheyne-Stokes type. As a rule, uræmic fits want the turgid purplish countenance and asphyxial character of true epilepsy—the face in uræmia being nearly always deadly pale and the breathing easy.

Dr. Richardson relates the cases of two children poisoned by belladonna berries, in which the symptoms closely resembled uræmic coma sequential to scarlatina. The insensibility was complete, and the pupils strongly dilated. The examination of the vomited matters and of the urine furnishes, in such cases, the best means of diagnosis.

In all cases of convulsions or insensibility from doubtful causes, the urine should be forthwith examined, and, if necessary, withdrawn by catheter for that purpose. It must not, of course, be forgotten, that sanguineous apoplexy is a not unfrequent occurrence in chronic Bright's disease, as in the case of J. H. before related (p. 428).

Uræmic coma and convulsions may prevail separately; but much more commonly the attacks are of a mixed character, and combine several or all the phenomena just enumerated.

As a rule, both the quantity of urine and the excretion of urea diminish notably at the period immediately preceding a uræmic attack. Sometimes, however, very great scantiness of urine, or even total suppression (in acute Bright's disease) may exist without evoking any uræmic symptoms. In a case of scarlatinal dropsy related by Biermer, complete suppression of urine continued for 5 days without uræmia; then followed a further period of 4½ days in which urine was secreted, but only in the scantiest proportions (a few teaspoonfuls a day), and yet no uræmia. At the end of this second period, the urine began to flow abundantly for a short time, and then again became scanty. Three days later uræmic coma set in, followed by convulsions, which proved fatal.

If the patient recovers from the coma, various mental symptoms, such as mania or melancholia, may show themselves.[1]

Uræmic *vomiting and diarrhœa* are common phenomena of Bright's disease. The vomiting which occurs in that disease is not, of course, always uræmic. The digestive functions are notably impaired throughout the complaint, and a heavy or indigestible meal may at any time be rejected, as in dyspeptic states from other causes. When the vomiting is really uræmic it takes place without reference to the nature of the contents of the stomach, and is oft-repeated or uncontrollable; the vomited matter is a watery fluid, either distinctly ammoniacal to the smell, or (if acid) evolving ammonia freely when caustic potash is added thereto. The alvine dejections are similarly characterized when due to the same cause.

Paroxysms of dyspnœa belong to the least frequent forms of uræmic disturbance—if indeed such attacks have at any time a genuine claim to the designation uræmic. Fournier cites some cases of this kind. Only the following somewhat doubtful example has fallen under my observation. The case is further remarkable on account of a transitorily ammoniacal state of the urine.

W. R. S., a railway porter, æt. 58, was admitted into the Manchester Infirmary, December 6, 1860. He was a stoutly made man, who had led an intemperate life. He had been remarkably healthy, and had scarcely ever lost a day's work. Two months before admission, his legs began to swell and then his face. He continued to follow his employment, though with difficulty, until two days before his admission.

On admission, there was a general anasarca of moderate degree; pallid features; enlarged heart; copious urine, scantily albuminous, with an abundant deposit of granular and transparent casts, and renal epithelium. Neither cast nor epithelium showed any signs of fatty degeneration. The urine was sometimes highly acid and deposited urates; at other times it was highly ammoniacal when voided.

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[1] Wagner. Ziemssen's Cyclopæd., 3d edition.

On December 27th there occurred a sudden and most intense paroxysm of difficulty of breathing, which threatened suffocation. It resembled in every respect a paroxysm of spasmodic asthma, and lasted five hours. It then passed away, and did not return again with the same intensity; though slighter attacks of a similar nature occurred on two other occasions.

On January 11, 1861, repeated vomiting took place; there was also a severe cough with a watery expectoration and increasing weakness. Somnolence then set in, which gradually passed into coma and proved fatal in three days.

The *autopsy* showed hypertrophied *left ventricle;* thickened mitral valve; abundant loose vegetations on the aortic valves. The right auricle and ventricle were filled with a firm voluminous yellow fibrinous clot. With the exception of dense œdema of the inferior lobe of the left lung, the *respiratory* organs presented nothing abnormal. The *kidneys* were found granular with commencing atrophy. The lower urinary passages were quite free from disease.

The ammoniacal state of the urine persisted in this patient for several successive days; and although the secretion was so charged with carbonate of ammonia that it had a pungent smell, and effervesced freely with acids, when quite fresh, the patient experienced no pain or uneasiness about the bladder nor during the act of micturition—which was not unduly frequent. The urine contained no pus. The ammonia in this case must have been derived directly from the blood, and not produced, as is usual in ammoniacal urines, by transformation of urea in the lower urinary passages. Such an occurrence betokened a free generation of ammonia in the blood; there were no uræmic symptoms on the days when the urine was ammoniacal. Did the elimination of ammonia by the kidneys stave off uræmic accidents?

THEORIES OF URÆMIA.—The absence of anatomical lesions in the brains of persons who die of uræmic coma and convulsions has constrained pathologists to look elsewhere for the determining cause; and by general agreement it has been assumed that that cause consists in certain alterations in the composition of the blood, from the accumulation in it of the excrementitious matters (and the products of their decomposition) which, in the healthy state, are removed out of the body by the kidneys. The blood thus poisoned is no longer capable of ministering to the tranquil and healthy operations of the nervo-muscular system, and engenders the various abnormities of motion and sense which have just been described.

But pathologists have not been content with this general appreciation of the matter, and have striven to trace the phenomena to the presence of some one, or the derivatives of some one, of these excrementitious substances in the blood. It will not be necessary in a practical work like the present to enter fully into the controversies—still undecided—which have arisen on this subject. It will suffice to indicate the different views which have been enunciated; and to express my own conviction, after a careful review of the observations and experimen adduced on all hands, that none of the exclusive theories of uræmia has made good its claim to acceptance.

Hammond and Richardson, following the original notion of Willis, contend that the special poison in these cases is urea.[1]

Frerichs and many more recent observers maintain, on the other hand, that urea is itself innocuous; that it may be injected into the veins of animals without detriment; that the mischief in uræmia arises from the transformation of the urea accumulated in the blood into carbonate of ammonia, and that the carbonate of ammonia so generated is the immediate excitant of the nervous symptoms. Frerichs upholds this doctrine by two propositions which he claims to have proved, namely: (1) that carbonate of ammonia invariably exists in the blood of uræmic patients, and in that of animals rendered uræmic by removing their kidneys, and can even be discovered in their expired air; (2) that carbonate of ammonia injected into the veins of healthy animals produces fits of convulsions with intervening pauses of coma, exactly resembling genuine uræmic attacks.

Treitz suggested a modification of this view. According to him, urea is not transformed in the bloodvessels, but is first vicariously excreted into the alimentary canal; here it is speedily converted by the gastro-intestinal mucus into carbonate of ammonia; the carbonate of ammonia so formed is then absorbed into the blood, and produces its poisonous effects. That urea is excreted by the intestines in Bright's disease is undoubted, and its swift conversion into carbonate of ammonia has been proved experimentally by Bernard. This theory of Treitz furnishes at least a rational explanation of uræmic vomiting and diarrhœa, and of the presence of the volatile alkali in the matters so discharged.

Since the theory of Frerichs was first promulgated, however, it has been ascertained by Richardson and Hammond that ammonia naturally exists in the blood of healthy animals; and all subsequent observers (with the sole exception of Petroff) have failed to discover in the blood of animals rendered uræmic by the removal of their kidneys any larger amount of ammonia than exists in the healthy state. It has been likewise shown that other substances than carbonate of ammonia (chloride of sodium, urine, and urea) are capable, when injected into the blood, of evoking comatose and convulsive phenomena.

The subsequent experiments of Oppler, Schottin, Perls, and Zalesky, seem to have given the *coup de grace* both to the ammonia and to the urea theories of uræmia; and they indicate, in a very clear manner, that uræmic manifestations depend mainly and essentially on the accumulation in the blood and tissues of those primary products of tissue-metamorphosis (creatine, creatinine, and other extractives), which, in a later stage of histolysis, are converted into urea and uric acid.

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[1] Richter found that solutions of urea (30 per cent.) applied directly to the sciatic nerves of frogs, produced only slight and uncertain convulsions of the muscles, far inferior to those produced by a solution of common salt. A saturated solution of urea produced no convulsions at all. (F. Ritcher, Inaug. Diss.: Erlangen, 1860.)

Cuffer[1] has recently asserted that the uræmic symptoms are due to destruction of the red blood-corpuscles. This may be produced by the various excrementitious matters which accumulate in the blood. He does not, however, reject the idea that the symptoms may, in certain cases, be due to spasm of the cerebral vessels, which view Dr. Hughlings Jackson maintains.

Feltz and Ritter[2] have asserted, that the poisonous substances accumulated in the blood are not the above-mentioned excrementitious matters, but potash salts.

Inquirers into the theory of uræmia may be reminded of the remarkable absence of coma and convulsions—the most common features of clinical uræmia — in cases of obstructive suppression of urine. The cases related in Chap. I., Section 5, of the present work show incontestably that fatal suppression of urine from blocking up the ureters generally runs its course without the usual symptoms of uræmia as witnessed in Bright's disease.

Dr. O. Rees believes that the tenuity of the blood in Bright's disease is not without influence in the production of the cerebral symptoms. Traube still further developed this idea. He contended that the watery state of the blood predisposes to interstitial transudations; that the hypertrophy of the left ventricle increases enormously the lateral pressure in the arterial system; that when, from any cause, a still further increase in the tenuity of the blood-serum occurs, serous transudation takes place through the cerebral capillaries, and gives rise to œdema of the brain. This œdema causes compression of the minute cerebral vessels, and determines an anæmic state of brain, and thereby uræmic convulsions and coma. He is further of opinion that the symptoms are of a comatose character when the œdema and anæmia affect the hemispheres, and convulsive when the central ganglia are the parts affected.

Jaccoud, in his "Pathologie Interne" (vol. ii. p. 443, 6th edition), asserts that the uræmic symptoms may in various cases be explained by several of the above-mentioned conditions, such as ammonia, creatinæmia, and Traube's œdema and anæmia of the brain.

Dr. Mahomed, again, has found small hemorrhages in the cortex of the brain, and these he believes to be the cause of the symptoms.

None of these theories, considered exclusively, explains satisfactorily the protean phenomena of uræmic intoxication, as witnessed at the bedside. The subjects of Bright's disease suffer under a deep abnormality in the composition of the blood and tissues. The blood is unnaturally watery and poor in albumen; the blood and tissues are unnaturally charged with the primary histolytic products (creatine, extractives, etc.), and with excrementitious urinary compounds (urea and uric acid,) perhaps also with the products of the decomposition of some of these. This state appears to induce in the nervous centres a proneness to sudden disorder and loss of equilibrium. A crisis may at any moment be brought about by an exaltation of one or several of the disturbing elements, or by a supervention of some new and different cause of irritation (hysteria, menstruation). A similar hypersensitive state of the nervous system prevails naturally in early life; and an irritation which would be of no moment in an adult (teething, worms, embarrassed digestion, cutanous irritation, etc.), suffices, in an infant, to awaken convulsive and comatose phenomena closely resembling those of uræmia.

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[1] Thèse de Paris, 1877.

[2] De l'urémie expérimentale, 1881.

DIAGNOSIS AND PROGNOSIS.

Diagnosis.—Under ordinary circumstances, chronic Bright's disease presents symptoms, and a condition of urine, so characteristic that it can scarcely be confounded with any other malady. Even when dropsy is absent, a persistently albuminous state of the urine, apart from heart disease, hardly belongs to any other condition.

Temporary albuminuria, as we have seen (see Congestion of the Kidneys), occurs occasionally under a variety of inflammatory and febrile conditions, without structural changes of any importance in the kidneys. These cases differ from Bright's disease in the absence of dropsical effusion; the quantity of albumen is also generally very small; the excretion of urea is natural or even excessive instead of being diminished. When defervescence occurs, it is speedily followed by the total disappearance of albumen.

The real diagnostic difficulties lie: (*a*) in distinguishing acute and curable cases from chronic confirmed ones; (*b*) in determining the precise anatomical changes going on in the kidneys; and (*c*) in detecting the disease when it is encountered masked by an inflammatory complication or a uræmic paroxysm.

(*a*) The case must be considered as belonging to the chronic and confirmed class, if the disease had crept on insidiously, or if it be found complicated with chronic phthisis, caries, long-continued suppurations, constitutional syphilis, enlarged liver or spleen, or hypertrophy of the left ventricle.

If the invasion have been acute, and the albuminuria still linger after the abatement of the febrile symptoms, time becomes a necessary element in the diagnosis. With every day that passes by without diminution of albumen in the urine, the fear grows stronger that the disease has become confirmed. In such a conjuncture the character of the urinary deposit supplies important information. If the epithelial elements and blood corpuscles continue to be freely discharged, and no, or only trifling, signs of fatty changes appear in the renal derivatives, there is good reason for confidence that the observer has to do with the declining stages of an acute disorder. If, on the other hand, albumen persist in considerable quantity after the pyrexia has passed away, and after blood has ceased, or almost ceased to appear in the urine, it is probable that the disease has lapsed into a chronic and confirmed state; and if, in addition to these untoward signs, the deposit exhibit marked fatty change, that probability becomes a certainty.

It must not be forgotten that patients suffering from chronic Bright's disease are subject to occasional febrile exacerbations, in which the urine becomes scanty, high colored, and perhaps bloody. Such exacerbations are liable to be confounded with the acute disorder; and when there are no clear indications of chronicity in the previous history, in the character of the renal derivatives, or in the coexistence of complications, the differential diagnosis of the two conditions may be quite impracticable until the lapse of time shall have cleared up the ambiguity.

(*b*) For the differential diagnosis of the different types of degeneration going on in the kidneys, the reader is referred to the synopsis of distinctive symptoms furnished in the first section of the present chapter.

(*c*) When the case comes under notice for the first time, masked by an inflammatory complication (pneumonia, endo- or peri-carditis, etc.), a clew to the primary disorder must be sought in the previous history of the case, and in the associated symptoms. When dropsy (or the history of any) is absent, the primary renal disease is apt to be overlooked, and the case regarded as one of simple inflammation of the organ affected: the urine (in such cases) assumes a febrile character, urea becomes abundant in it, and its specific gravity rules high. If, under such circumstances, the quantity of albumen in the urine be but small, the absence of Bright's disease may be counted on; but the converse deduction is not invariably warranted. In pneumonia I have seen the urine for some days "highly" albuminous without, as the sequel showed, the existence of any renal degeneration. In pleurisy and pneumonia (and especially the former) the simultaneous implication of the two sides furnishes a strong presumption (supposing the urine be albuminous) that the inflammation is not simple, but secondary to renal disease. The existence of cardiac hypertrophy without valvular disease, or of notable anæmia, also favors the supposition of Bright's disease.

The differential diagnosis of uræmic coma and convulsions has been already pointed out (see p. 432).

The absence of casts in an albuminous urine gives no security against the existence of renal degeneration; indeed, this absence is generally more apparent than real. When the casts are few in number, and small, they subside very imperfectly, and are apt to escape detection, even with the most careful examination.[1] In other cases the absence of casts is only temporary; and I have known it most absolute in some of those sad hopless cases, where the renal disease is the ultimate upshot of an intractable strumous or syphilitic cachexia.

PROGNOSIS.—The prospects of a patient suffering from confirmed chronic Bright's disease are exceedingly gloomy. The textural changes in the kidneys are of a kind that do not admit of reparation. The Malpighian bodies become enveloped in an exudation of low plastic material, of which the only tendency is to progressive contraction, and the tubuli are either blocked up with fibrinous plugs or shrivelled into useless fibres. The gland is not, however, equally affected throughout all its parts, and the less injured portions carry on, imperfectly, the depurative functions. As the sounder portions become more and more involved—and there is an almost inevitable, though slow, tendency to this—the work done by the kidneys grows less and less, and the blood is more and more contaminated with histolytic and urinous elements, until at length a limit is approached, which is incompatible with life. Long before this extreme limit is reached, however, death is brought about in a large number of cases by one or other of the numerous complications to which the subjects of renal degeneration are obnoxious.

In certain more favorable cases the structural changes cease to advance, the dropsical effusions (if any existed) are absorbed, and the condition of the patient remains stationary, perhaps for months, perhaps for years; and he may be able, with proper precautions, to prolong existence in fair comfort, and even to pursue light avocations, for very considerable periods of time. Cases protracted to five or six years are not uncommon, and a few instances are recorded in which the patient has survived for ten, fifteen, or even twenty years.[2] The tenure of life under these circumstances is exceedingly precarious, and an imprudent indulgence or exposure may bring life, in a few hours or days, to the verge of destruction; the patient walks, as it were, on a slumbering volcano, which may at any moment awaken its fires with a fatal explosion.

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[1] Some years ago I was consulted by a medical man who was suffering from persistent albuminuria. He himself, and another practitioner, who was well accustomed to such inquiries, had failed, after repeated examinations, to detect a single cast in the urine. The specimen of urine sent to me was set aside for twelve hours in an appropriate urine glass. At the end of that period I could not, after long searching, discover a single cast. Next day, however, the urine deposited an abundant sediment of very minute uric acid crystals. On again searching for casts I found several without difficulty—the precipitation of the uric acid had carried them down.

[2] The protracted survivorship of some cases of chronic Bright's disease is very remarkable—in some rare cases the disease does not prove fatal at all. Possibly in these cases only one kidney is affected—its fellow remaining sound. That such a possibility exists may be inferred not only from the great difference in the amount of disease sometimes found in the two kidneys after death, but more certainly from such a case as that of Dr. Moxon, in which one kidney was in an advanced stage of degeneration, while its fellow was normal. Path. Soc. Trans., v. xix. p. 268.

But although the final prognosis in chronic and confirmed cases is thus unfavorable, the immediate prospects of the patient may be fair, and there is still hope that by judicious management amelioration of the more distressing symptoms may be brought about, except in the ultimate stages of the disease, the dyspeptic symptoms, the irregularities of the bowels, the dropsical accumulations, and the bronchial catarrh, may be combated with good probability of success. The following example is a striking illustration how near an apparent cure the subsidence of the symptoms may proceed, even from an apparently desperate extremity.

Mr. B., a designer, of sober habits, æt. 38, consulted me, May 9, 1862. He was suffering from great and general anasarca with ascites. The urine was scanty and intensely albuminous. There was an abundant deposit of tube-casts and renal epithelium. These structures exhibited the appearances of advanced fatty degeneration.

The patient stated that the dropsical symptoms had existed a twelvemonth, and had come on gradually—first in the feet, then in the face—without known cause.

A fortnight after, I was requested to see Mr. B. at his own house. He was then confined to bed; the swellings had considerably increased; the legs were tense, and incapable of being moved from excessive œdema; the peritoneal effusion was very great. There was severe dyspnœa (orthopnœa) and frequent vomiting. The urine was almost suppressed.

Taking into consideration the state of the urine, the character of the deposit, and the time the disease had already existed, together with the threatening gravity of the general symptoms, it seemed hardly possible that the patient could rally to anything like seeming health; and yet this took place. Compound jalap powders were administered freely, and blanket-baths applied daily. Copious watery motions were produced; the legs burst, and an immense quantity of fluid drained away. Improvement went on steadily in the course of the ensuing month; in September the dropsy and ascites had nearly disappeared. A specimen of urine (of which the flow was copious) was sent to me at this time for examination. It was only slightly albuminous, and after a diligent search I failed to detect any casts.

I did not see the patient after this; but Mr. Briggs, with whom I saw the case, informs me that shortly after, the patient went to Wales, where he continued to improve; the œdema disappeared almost entirely; the appetite returned; and the strength was so far restored that he was able to walk fifteen miles in a day.

On his return to town, however, the dropsical symptoms reappeared and increased; and a cough set in, accompanied with purulent expectoration. The pulmonary symptoms gradually advanced, and he died in September, 1863, about eighteen months after I had first seen him.

The favorable and unfavorable signs in Bright's disease have relation to the state of the skin, the duration of the disease, the degree of deviation of the urine from its natural quantity and composition, and the existence of complications.

The signs which indicate that an unfavorable termination is not far distant are: obstinate dryness of the skin, the urine, which had previously been abundant, becoming steadily scantier without proportionate increase in the specific gravity, evidence that the disease has existed some years, repeated recurrence of uræmic phenomena, excessive serous effusion, excessive cardiac hypertrophy, a persistently feverish state. Speedy death is indicated by the breaking forth of pneumonia or pericarditis, by suppression of urine or uncontrollable vomiting and diarrhœa. The absence of these signs may be construed in a favorable sense, as indicating a stationary state, and the probability that the final issue may be yet far distant.

An excessive proportion of albumen in the urine, although a proof of the activity of the morbid process, and therefore a sign of evil augury, is not necessarily prophetic of impending death. In a case which I saw with Mr. Stephens, the urine, which was examined almost daily, became constantly solid on boiling, for a period of more than two months. During this period the patient's condition was stationary; he was then seized with pneumonia, of which he speedily perished.

TREATMENT.

In the management of cases of confirmed Bright's disease, three objects are to be especially aimed at, namely: (*a*) to hinder the further extension of the structural changes in the kidneys; (*b*) to prevent the occurrence of uræmic and inflammatory accidents; and (*c*) to palliate or remove certain threatening or burdensome symptoms—anæmia, dropsy, dyspeptic and uræmic phenomena, etc.

To fulfil the first indication, the conditions under which the complaint originated must be carefully traced out, and the patient removed as completely as possible from their further influence. In some instances this is practicable; as when the disease follows intemperance or long-continued exposure to wet and cold. In protracted suppurations, necrosis, caries, joint disease stricture of the urethra, and old vesical inflammation, the possibility of the development of renal degeneration should be kept in view by the surgeon, and should have weight in considering the propriety of operation. In all such affections the condition of the urine should be narrowly watched; and the first appearance of albumen is a warning that the opportunity for operative procedures is slipping away, never to return.

There is no evidence that local counter-irritants of the severer class—issues, setons, moxas, etc.—applied over the kidneys, exert any good effect; and the ulcerations they sometimes leave are apt to prove intractable. Mustard poultices, tincture of iodine, or dry cupping may be applied when the loins are the seat of aching pain; but their influence on the renal lesion is probably *nil*. Blisters are inadmissible on account of their specific irritating effects on the urinary system.

The patient should be habitually clothed in flannel, both limbs and trunk; and the activity of the skin should be further encouraged by moderate walking or carriage exercise, and the occasional use of warm baths and frictions of the surface. The bowels should be opened at least once daily, and the diet should be light and nutritious. Milk agrees well with the majority of this class of patients, and may be freely partaken of.[1] Two or three glass of claret or hock daily, or a glass of sound beer, are permissible: but the stronger wines and all spirits agree, as a rule, badly, and should not be allowed unless special circumstances imperatively call for their administration.

Medicinal agents of roborant character should be exhibited from time to time—but especially preparations of iron. I have been in the habit, when the secondary symptoms or complications do not call for special treatment, of contenting myself with giving 15 or 25 drops of the muriated tincture of iron in a wineglass of water night and morning—combined, in cases of strumous affinities, with cod-liver oil. If the tincture produces headache or disturbs digestion, some other chalybeate must be substituted—the citrate of iron, citrate of iron and quinine, the syrups of the phosphate or the iodide of iron, the saccharated carbonate or the ferrum redactum. One or other of these preparations can generally be made to agree. It is important to get patients with chronic Bright's disease to take iron, for saturation of the system with iron is the best safeguard against the profound anæmia which is a fertile source of danger and distress to the sufferers from chronic renal degeneration.

Are there any medicinal substances capable of exercising control over the quantity of the albumen lost by the urine? Exact observations do not give an affirmative answer to this question, though a certain reputation has been gained by the mineral acids (especially nitric acid), iodide of potassium, tannin, and gallic acid. Dr. Parkes exhibited large doses of tannin and gallic acid without producing any diminution of albumen. I have in a number of cases used gallic acid for a period of many weeks, but could not convince myself in a single instance that it had any favorable influence on the excretion of albumen, and in some instances it occasioned serious gastric disturbance. Oppolzer has recommended alum, and the trials of Heller support this recommendation.

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[1] Drs. Sparks and Bruce have made some very careful observations on the influence of diet in a case of Bright's disease They found that an absolute milk diet, or an absolute non-nitrogenous diet, effected a reduction in the amount of albumen excreted. (Med.-Chir. Trans., 1879, p. 243.)

Knowing as we do that persons with albuminuria and degenerated kidneys may preserve passable health for years, so long as digestion is good, the blood not too impoverished, and the complications kept away, the practitioner is not justified in interfering too actively when this stationary condition is maintained: he should confine himself to the enforcement of sound hygienic rules and preventive measures. The patient should be made clearly to understand that he is to treat himself as a valetudinarian; and that in his clothing, his eating, drinking, exercise, and general mode of life, he must go by rule, as the sole condition of not running the most fatal risks.

The most effective means of combating the *dropsical effusions* are hydragogue cathartics and warm baths. For general use there is no hydragogue superior to the compound jalap powder with an additional quantity of the bitartrate. It acts quickly, and procures two or three copious watery stools. The objection to its use is the nausea and sickness which it too often occasions. To diminish this inconvenience as much as possible, an active dose (for an adult ʒiij of the bitartrate, and 15 or 20 grains of jalap corrected with a little ginger) should be administered early in the morning twice or thrice a week. The operation of the medicine passes over in a few hours; and the patient has leisure to recruit himself in the intervals between the doses. This proceeding is less harassing than to keep up a continued purgation of less activity.

Christison speaks in high terms of gamboge, which he employed in doses of 5 grains, sometimes 7, and very rarely 9 grains, every second day, or in urgent circumstances every day. To prevent griping he had it finely pulverized with half a drachm of the bitartrate of potash. Colocynth, scammony, and elaterium have also been employed on divers hands. When the serous accumulation is very threatening, and immediate effects are demanded, no remedy is superior to elaterium. It may be given in doses of one-sixth or one-fourth of a grain every three or four hours, until three evacuations have been obtained. In the employment of purgatives, it must not be overlooked that exhausting diarrhœa sometimes occurs spontaneously in the later periods of the disease, and that the use of drastics has been known to originate this untoward symptom.

It is necessary, therefore, to watch the action of these evacuants, and to desist from their use immediately if the diarrhœa shows signs of proving intractable.

Warm baths are unquestionably the most effective of diaphoretics; they not only promote cutaneous transpiration, but often increase the secretion of urine at the same time. They may be applied in all their varied modifications—warm water, hot air, steam, or the blanket-bath. When one modification fails another may succeed. Dr. Liebermeister describes as highly effective, a method of applying the warm water bath, by which the temperature is gradually raised after the bather is immersed. When the patient first enters the bath the temperature is 98°; it is then gradually raised by the admission of warm water to 108°; after remaining in the bath about thirty-five minutes, the patient is packed in hot blankets.[1]

Unpleasant consequences—headache, suffusion of the face. unwonted heat of skin—occasionally follow the use of warm baths, and may even necessitate their abandonment. Generally these inconveniences diminish as the remedy is repeated; and after a few trials patients with chronic renal disease nearly always take their baths with pleasure as well as with advantage.

Of pharmaceutical diaphoretics, Dover's powder, James's powder, and liq. ammon. acetatis have been chiefly used: their effect is very uncertain.

Diuretics are of much inferior value to purgatives; but when there exists a tendency to spontaneous diarrhœa or to severe gastric derangements, we are constrained to abandon the latter for the former. My experience of diuretics has not given me a high opinion of their efficacy. The testimony of authors on their utility is conflicting. In judging of their effects, some observers have not sufficiently considered that a spontaneous diuresis is the normal outgoing of acute renal dropsy tending to recovery; and that in patients with contracting kidneys profuse diuresis is an ordinary feature of the quiescent state in the middle periods of the disorder, so that when the urine becomes scanty and the dropsical effusions increase during an intercurrent febrile exacerbation, the reëstablishment of the diuresis and the diminution of the anasarca on regaining the quiescent condition, must not be too hastily attributed to the diuretic which chanced to be employed pending the pyrexial attack.

Diuretics of the most opposite classes have been recommended by different writers. Bright, who had but slight confidence in diuretics, was in the habit of prescribing uva ursi and pyrola umbellata. Christison relied on digitalis combined with cream of tartar. Rayer perceived little advantage in digitalis or squills, and he found that they almost always, at length, deranged the stomach. Horseradish tea,[1] according to his experience, offered of all diuretics the best chance of success. Spruce beer is a much more agreeable beverage, and its diuretic action is probably not inferior. My late colleague, Dr. Eason Wilkinson, has repeatedly obtained good results from its use; and on his recommendation I have tried it myself in a number of cases, with favorable effects; it agrees well with the stomach, and quenches the thirst which not unfrequently torments patients with Bright's disease, more effectually than any beverage I know.

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[1] Prag. Vierteljahrschr., 1861.

Tincture of cantharides was employed by Dr. Wells in seven cases, in doses of 30, 50, or even 60 drops per day, with good effect in five. Rayer also reports well of it in some cases; but he adds, not without reason, "it is an uncertain remedy, which might be dangerous in inexperienced hands."

I have tried in my own practice dandelion, broom-tops, and belladonna with unsatisfactory results.

When other means of evacuating the dropsical effusions fail, and the tension of the integuments threatens erythema and gangrene, there is yet a resource in acupuncture or incision of the legs. This rapid and easy method has the disadvantage that, unless stringent precautions are taken, the punctures are liable to become the focus of erysipelatous inflammation, which may spread and pass into sphacelus, with disastrous consequences. This mishap is quite as likely to follow needle punctures as incisions, and after trial of both plans, I prefer the latter. One or two cuts with a lancet should be made lengthwise in the calf of the leg, or one of them may be placed on the dorsum of the foot. The incision should be three-quarters of an inch long, and penetrate fairly into the subcutaneous tissue. To prevent erysipelas the following directions should be carried out; the incised member should be wrapped in hot, moist flannels; these should be changed frequently—at first every two or three hours. At every change, the legs, and especially the incised parts, should be thoroughly sponged with warm water, and the flannels which are soaked with the discharge should be completely cleansed before they are reapplied. Traube recommends that the incisions be frequently washed with chlorine water. Under such precautions this treatment may be carried out with safety—always with great relief at the time, and sometimes with prolonged advantage.

Dr. Southey[2] has introduced, in the form of his capillary drainage-tubes, a most convenient apparatus for removing the watery accumulations in the limbs. These tubes avoid to a great extent the danger of erysipelas.

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[1] Half an ounce of the root infused in two pints of water was the dose with which Rayer usually began; this was gradually increased to one or one and a half ounces. The dried root makes a less acrid infusion than the fresh root, of which a smaller quantity must be used (Mad. d. Reins, ii. p. 152).

[2] Lancet, 1877, I. p. 649.

In those cases which are characterized by a copious flow of urine (contracting kidney), dropsical effusions, if present at all, are usually slight and partial; and their existence depends chiefly on the watery state of the blood, and the lowered tonicity of the bloodvessels. In these cases, diuretic and cathartic remedies avail little to diminish the œdema; better results are obtained by ferruginous preparations, tonics, and mineral acids. If the patient's general health can by these means be effectually improved, the serous effusions will not delay their disappearance. It is in cases of this class likewise that change of air, or even a sea-voyage, may be recommended, provided always that the disease be not too far advanced.

The treatment of *bronchial catarrh and secondary inflammations* requires to be undertaken with a remembrance of the primary mischief. Mercury and bloodletting are inadmissible—the former (unless in the most guarded way) on account of the peculiar susceptibility of the system in Bright's disease to mercurial preparations, the latter on account of the deep undermining of the strength which has already taken place. Internal antiphlogistics—aconite, digitalis, and antimony—may be freely used; also external applications—chloroform epithems, hot poultices, dry cupping, etc.

The *dyspeptic* symptoms are readily controlled in the early periods by a careful revision of the diet, and the use of vegetable bitters, prussic acid, and antacids.[1] When obstinate vomiting of uræmic origin sets in, it is very difficult to subdue; creasote, morphia, and ice permitted to melt in the mouth, are the most effective remedies. Diarrhœa of similar origin must be combated by acetate of lead, opium, and sulphuric acid.

When *uræmic* symptoms show themselves, renewed efforts should be made to increase the flow of urine, and to awaken the vicarious activity of the skin and intestines by the measures already described. If coma and convulsions have actually seized the patient, further energetic action is demanded. Frerichs, consistently with his view that carbonate of ammonia is the actual poison in these cases, recommends a treatment designed to neutralize the free ammonia, and reduce it to a state of innocuous combination. He directs chlorine (which may be inhaled in the gaseous state or taken dissolved in water) and the vegetable acids to be taken internally, the body to be sponged with vinegar, and vinegar to be used in injections.

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[1] Dr. Lauder Brunton has found arsenic of great service in cases of Bright's disease, where dyspeptic symptoms are prominent.

During the convulsive paroxysm, chloroform inhalation is the most prompt and ready means of controlling the spasms. When the uræmic paroxysm begins with drowsiness and gradually passes on to insensibility, or when convulsions occur only as breaks in a continuously comatose condition, chloroform affords no prospect of relief. Chloral, especially if administered by the rectum, is of great service in controlling the convulsions.

At the time when Bright, Christison, and Rayer published on this subject, everything in the shape of an apoplectic or convulsive seizure was the signal for immediate and copious venesection; it is not surprising, therefore, to find in the cases they recount that free and repeated bleedings are almost invariably chronicled in the next sentence to that announcing the advent of the uræmic paroxysm. Dr. Richardson has recently advocated the same plan. The immediate effect is, unquestionably, in a large number of cases, to relieve the insensibility; consciousness sometimes returns as the blood flows. But the indiscriminate use of this powerful remedy is the surest way to bring it into ultimate disrepute. A distinction should be drawn according as the renal disorder is acute or chronic and according to the strength and general condition of the patient. It must be borne in mind that an impoverished and watery state of the blood is an effective factor in the generation of uræmic phenomena, and that a bloodletting, though it may relieve a present attack, increases the predisposition to future attacks. In the uræmic coma of acute Bright's disease, and in certain classes of puerperal eclampsia, the blood is as yet not materially impoverished, and the type of renal mischief is one that gives full hope of eventual recovery, while the attack itself is of extreme danger. In these, venesection—free, and even repeated—is decidedly and urgently demanded. But the matter stands otherwise when the renal mischief is chronic and incurable. The attacks themselves are not so imminently dangerous as when occurring in the acute form of the disease, patients frequently survive repeated uræmic paroxysms without the aid of venesection;. the blood is commonly thin and poor; and, lastly, there is not any prospect of ultimate recovery. Under these circumstances loss of blood is more likely to shorten than to lengthen life. Further, as Christison remarks (speaking of advanced renal mischief), when the torpor becomes considerable the removal of blood seems of little or no use. In some of the cases reported by Bright, death occurred from coma on the very day of free and repeated venesection. I can only conceive of two contingencies in which withdrawal of blood, in quantity, is justifiable in chronic renal disease; one is, when coma comes on rapidly in a person whose constitution is not, as yet, seriously deteriorated, and whose prospects of life (abstracting the uræmia) may extend to many months or some years; the other is, when there is a necessity for temporary restoration of the faculties paramount to the general chance of prolonging life.

When the comatose symptoms come on gradually, the measures before enumerated should in every case take precedence of bloodletting.

Of late years, a therapeutic agent, most valuable in cases of uræmia, has been introduced, in the form of pilocarpin, the alkaloid of jaborandi. When a dose of the hydrochlorate of pilocarpin (¼ to ½ grain) is injected under the skin, profuse sweating follows in a very short time, much to the relief of the uræmic symptoms. The drug is also of use in dropsical states. It is prone, however, to cause symptoms of collapse, which must be guarded against by the use of stimulants.

CHAPTER V.

SUPPURATION IN THE KIDNEY—RENAL EMBOLISM.

Purulent formations in the substance of the kidney arise under three conditions, namely: (1) phlegmonoid inflammations ending in circumscribed abscess; (2) multiple abscesses from purulent infection; and (3) occasionally from embolism apart from pyæmia.[1]

It is necessary to distinguish between abscess situated in the substance of the kidney, and purulent distention of the pelvis and infundibula, with ultimate sacculation of the organ (pyonephrosis). These two conditions were confounded under the common name of "abscess of the kidney," until Rayer pointed out the distinction between them.

1. Phlegmonoid Abscess is nearly always confined to one kidney. It may be due to external violence (blows or falls on the loins), or to inflammation and suppuration round a calculus or calculi lodged in the substance of the gland, or it may arise as a sequence to suppuration of the lower urinary passages. In the last-mentioned cases the abscess may be due to extension of the inflammation by continuity of tissue along the straight ducts of the pyramids; sometimes, however, the primary inflammation is confined to the bladder or urethra and no direct connection between the two foci of suppuration is evident. Such cases are difficult to explain. Most probably the view pro-

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[1] Dr. Johnson describes another form of "suppurative nephritis," in which pus is produced in the uriniferous tubes by transformation of the renal epithelium. This condition he found associated with the occurrence of "purulent casts" in the urine. I have frequently noticed casts of this character, but am disposed to explain their appearance differently. It is not very rare, in Bright's disease, to find, in the urine, cells attached to fibrinous casts with double or triple nuclei. But this is no more evidence of pus than the occurrence of the pale corpuscles in the blood (which are, anatomically, indistinguishable from pus corpuscles) is evidence of pus in the blood. When the renal cells proliferate rapidly, they assume very much the appearance of pus corpuscles, and display cleft nuclei.

The cases adduced by Dr. Johnson are quite inconclusive; the first (Case 28) was an example of Bright's disease complicated with boils; pyæmia followed and metastatic abscesses were found in the kidneys and lungs; the second case (No. 29) seems to have been an example of the mottled white kidney acutely developed—complicated with cutaneous erysipelas, but otherwise not unusual in its course. In the third case dropsy and albuminuria of sudden onset had come on in an intemperate and gouty man. Dr. Johnson found purulent casts in the urine, but after a while they disappeared, and the patient survived nearly a year. To apply the term suppurative nephritis to cases like the last two is likely to mislead, for they differ nowise clinically from typical Bright's disease.

pounded by Klebs,[1] that micrococci are the agents of propagation, is correct. In not a few cases, the suppuration in the kidney is merely a local manifestation of a general pyæmic condition starting in the lower urinary passages. An abscess formed in any of these ways may involve the whole kidney in destruction and convert it into a bag of pus.

An abscess of the kidney generally opens into the pelvis of the organ, and its contents are discharged by the ureter. This is by far the most favorable issue. Sometimes the pus works its way out in other directions; it may penetrate the tunica propria posteriorly, and be evacuated in the loins; and recoveries have taken place even after this event, though generally such cases prove ultimately fatal. A renal abscess, more rarely, bursts into the colon or duodenum, and is discharged by stool; or it penetrates into the peritoneal sac, causing rapid death; in still rarer cases, it has been known to push into the cavity of the thorax and be evacuated by coughing.

The symptoms of circumscribed abscess of the kidney are pain in the affected organ, fever, hæmaturia, successive (often regular) rigors; and, if the collection be sufficiently large, a fulness or fluctuating tumor is perceived in the renal region. When the abscess bursts into the infundibula there is sudden and copious discharge of pus with the urine—or, if it burst into the intestines, with the stools—followed by simultaneous subsidence of the tumor. In the absence of fulness in the renal region, or of signs of pointing in the loins, the diagnosis is necessarily uncertain. Perinephritic abscess is sometimes accompanied with hæmaturia, and other signs greatly resembling those of abscess in the renal substance.[2] Albuminuria is by no means a necessary symptom of suppuration in the kidney. It may be wholly absent, or the urine may contain a small quantity of albumen. When abscesses of the kidney form slowly in the course of suppurating disease of the lower urinary passages, they are usually unassociated with any special symptoms, and their existence may not be suspected until the autopsy.

Sometimes the contents of a renal abscess, instead of being evacuated, are gradually inspissated; the liquid parts of the pus are absorbed, and the residue is dried up into a putty-like mass containing shrunken pus corpuscles mixed with considerable quantities of phosphate and carbonate of lime. When an abscess dries up in this manner it may lie permanently latent, and give no further trouble. The destruction of one kidney in this way is compensated by enlargement of the opposite healthy organ, which is thus enabled alone to carry on the renal function.

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[1] Handbuch d. path. Anatomie, p. 654. The septic material may reach the kidney through the renal tubules, or, as pointed out by Mr. Marcus Beck, it may pass into the lymphatics surrounding the pelvis and straight tubes of the kidney, by means of breaches in the epithelial surface, and so become disseminated through the whole organ. *See*, also, a paper by Dr. Stevens, Glasg. Med. Journ., Sept. 1884.

[2] *See* a case by Todd. Clin. Lects. on Ur. Dis., p. 39.

TREATMENT.—When external violence has been so inflicted that there is reason to apprehend suppurative inflammation, the loins should be freely cupped or leeched; absolute rest and low diet should be enjoyed for several days, and the bowels kept open by emollient clysters. The same treatment should be followed in principle, but modified to suit the accompanying circumstances, if abscess is threatened from renal calculi.

When signs of pointing in the loins are recognized, they should be encouraged by warm poultices; and as the pus approaches the surface it should be evacuated, in order to forestal the danger of evacuation by the more dangerous channels of the peritoneum or thorax. Too often the original cause of the suppuration (impacted calculi or disease of the lower urinary passages) is irremovable, and the evacuation of one abscess is liable to be followed by the formation of others, which at length exhaust the patient.[1]

2. MULTIPLE OR METASTATIC ABSCESSES.—Secondary abscesses are sometimes found in the kidneys, as well as other parts of the body, after death from pyæmia. The kidneys are, however, less frequently the seat of such abscesses than the lungs and liver. In 2161 autopsies performed at St. George's Hospital, Dr. Chambers found pyæmic abscesses in the kidney 12 times; in the lungs, 106 times, and 22 times in the liver. The implication of the kidneys has been found in pyæmia from almost every cause—after amputations, lithotomy, lithotrity, and other surgical operations, gangrenous affections, carbuncle, glanders, variola, chronic suppurations, especially of the lower urinary passages. In four instances Dr. Chambers found the kidneys free from abscesses when the pyæmia had arisen from disease of the urinary passages themselves.

The primary disorder (pyæmia) is of such overwhelming gravity, that the renal lesion becomes by comparison unimportant. No symptoms are known whereby the existence of multiple abscesses in the kidneys can be predicated with certainty during life. A strong probability that such abscesses exist will arise if, with evident pyæmia, the renal regions are painful on pressure and a considerable quantity of albumen be discharged with the urine.

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[1] Illustrative cases of abscess of the kidney may be found (in addition to those indicated at the head of the chapter) in the following sources: Lancet, 1847, i. p. 335; 1853, i. p. 32; 1863, ii. p. 69; 1873, ii. p. 772. Med. Times and Gaz., 1854, i. p. 23; and ii. pp. 241, 343; 1874, ii. p. 632. Med. Gazette, vol. xix. p. 888; xxiv. p. 563; xxvii. p. 141; xlv. p. 252. Path. Soc. Trans., 1849–1850, p. 234; vol. v. pp. 178, 179; xiii. p. 131. Gaz. Hebd., 1863, p. 40. Dublin Hosp. Gaz., 1854, p. 147; and vol. i. p. 121. Med.-Chir. Rev. (1824–1834), new series, xii. p. 81; xix. pp. 159, 234. Med. Commentaries, vii. p. 41. Med. Facts and Obs., vi. No. 3. Encycl. d. Sc. Med., v. 54, p. 19. Wilson, Lects. on Dis. of Ur. Organs, Lond. 1821, pp. 281, 283. Bennet, Clin. Lects., 2d ed., p. 731. Ulrich, cited by Rosenstein, loc. cit., p. 287. Southey, Clin. Soc. Trans., 1869, p. 58. Burritt, Med. and Surg. Rep. of New Orleans, 1868, p. 520. Hough, Amer. Journ., 1870, p. 280, also Arch. Gén., 1869, p. 348.

Sometimes the secondary abscesses in pyæmia are almost confined to the kidneys, as in the following example:

On January 9, 1865, I examined the body of a man, æt. about 45, who had died in the Salford Workhouse, a day or two after his admission, of some obscure disease. The bladder was found thickened and contracted; the mucous membrane softened, red and congested, but not ulcerated. The ureters were dilated to the thickness of a large quill. The kidneys were enlarged to about twice their natural size, and riddled with hundreds of minute abscesses, the largest of which were about the size of a hoarse-bean and the smallest like pins' heads. They were distributed through the substance of the organs and on their surfaces, mostly aggregated into groups varying from the size of a sixpence to that of a florin. Each abscess was surrounded with a red inflamed areola. Some of those on the surface appeared thinned away almost to bursting, and in places, resembled a patch of herpetic eruption. Not one of these abscesses had opened into the infundibula; and if pus had made its way into the ureter from the kidney, it must have drained along the uriniferous ducts. On section it was seen that the little abscesses displayed a general tendency to range themselves end to end, in lines following the direction of the ducts of the pyramids.

There were about 8 ounces of urine in the bladder; it deposited, on standing, an abundant sediment of pus; it also contained a good deal of albumen—more than the pus accounted for. Careful examination failed to discover casts of tubes of any sort.

Both lungs contained a few secondary abscesses; there was abundant recent pleurisy on both sides. The right heart was somewhat dilated; otherwise the organ was healthy. The liver was healthy.

The pyæmia in this case appeared to have arisen from chronic cystitis.

The exciting cause of the abscesses is probably in every instance the presence of micrococci. Often the capillaries in the neighborhood are found crowded with micrococci, and no embolism can be discovered. In some cases, however, a small vessel is blocked by a portion of clot discharged from an inflamed vein, and, in the manner to be presently described, an infarct is produced which rapidly goes on to suppuration; the determining cause of pus-formation, even in this case, is most likely the irritation produced by the presence of microörganisms.

3. **Renal Embolism.**—It was well known to Rayer that rheumatic endocarditis was sometimes attended with the formation

of numerous deposits of a yellow color in the kidneys, which he considered to have the nature of plastic lymph. He described and figured such cases under the designation of "néphrite rhumatismale."[1] But it was not until the doctrine of embolism was worked out by Kirkes, Virchow, and others, that the real nature of these lesions was understood.

The loose fibrinous vegetations which beset the aortic and mitral valves in endocarditis are apt to be detached, and swept away with the current of blood into the arterial system, and to be finally impacted in some small artery in the brain, kidneys, or other part. But it is not solely in endocarditis that such masses are dislodged and carried away. The same may happen in atheromatous erosions of the valves and aorta; or a portion of the fibrinous lining of an aneurism may be detached and whirled away to the kidneys and other places.[2]

The effect of the lodgement of plugs or emboli from any of these sources in the arteries of the kidneys varies with their magnitude. Small emboli either do not produce any appreciable symptoms, or they merely occasion a dull uneasiness in the renal region; and their existence is only ascertained by inspection after death. But if one of the larger arteries is plugged up, not only does the occurrence produce symptoms referrible to the kidneys (sudden acute pain in the loin shooting down the ureter), but it may, under favorable circumstances, even be diagnosticated during life.[3]

In three cases of embolism described by Kirkes (in which death ensued from softening of the brain, consequent on obstruction of one of the main cerebral arteries, by a fibrinous plug derived from warty vegetations on the valves of the left heart), the kidneys were the seat of numerous masses of yellow deposit surrounded by red areolæ. An injection thrown into the renal artery did not penetrate in the least degree into these yellow patches. The further researches of Virchow and Beckmann have added some particulars to the description of Kirkes. The yellow spots are situated almost exclusively in the cortical substance; they are frequently wedge-shaped, with their bases bulging underneath the tunica propria, and their apices pointing toward the infundibula. They vary in size from a hemp-seed to a hazel-nut. At first they look like red hemorrhagic patches; in the centre of each there soon appears a yellow spot.[4]

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[1] Rayer, loc. cit., t. ii. p. 73. Atlas, pl. v.

[2] *See* the history of a case of aortic aneurism, by Murchison and Moore, in vol. xlvii. of the Med.-Chir. Trans., p. 129.

[3] Such a case is related by Traube, loc. cit., p. 77.

[4] From more recent researches, it appears doubtful whether the infarct of the kidney ever assumes the hemorrhagic form, except when occurring together with venous stagnation. Certainly, in the post-mortem room, it is nearly always met with in the form of a yellow mass. The reason of this is not clear.

This enlarges, and either softens in the centre (breaking down into a fatty débris, more rarely into genuine pus) or finally contracts into a cicatricial remnant, with destruction of the adjacent secreting tissue. It is necessarily a matter of extreme difficulty to demonstrate the existence of obstructions or plugs in the minute vessels at or near one of these yellow spots, and some doubt yet hangs over the demonstration. When one of the main branches is obstructed, the embolus is more easily discovered, and the appearances are somewhat different. The first effect of cutting off the arterial supply of a considerable section of the kidney is to produce intense hyperæmia of the surrounding parts, which results in rupture of the capillaries and effusion of blood into the surrounding tissue. In this way a wedge-shaped apoplectic area is formed, embracing the whole thickness of the organ. Cohnheim found that in similarly produced lesions in the frog's tongue, the current in the veins was reversed, and assisted in the congestion of the infarct.

As a rule, the effects of embolism in the kidney are of very slight clinical importance; they pass by, in the immense majority of cases, without recognition; their occurrence is always secondary to some much more grave primary disorder, which altogether dominates the prognosis and treatment. Sometimes the larger ones go on to suppuration, and, still more rarely, to gangrene.

CHAPTER VI.

PYELITIS AND PYONEPHROSIS.

Morbid Anatomy.—Inflammation of the pelvis and calices of the kidneys, or pyelitis, may be acute or chronic; it may involve the two sides, or be confined to one.

In *acute* pyelitis the mucous membrane is injected; sometimes minute ecchymoses dot its surface, and blood may be effused on it; the epithelium is more or less freely shed, and at a later period pus is formed. In rare cases, the surface is lined with false membranes (diphtheritic pyelitis) which are liable to be detached, and to block up the ureter.

In *chronic* pyelitis the mucous membrane has a dead-white color, sometimes gray, or slate; either it is not at all injected or it is traversed by dilated veins. The membrane is also thickened. and the pelvis and infundibula are dilated. This dilation, as it proceeds, encroaches more and more on the substance of the gland: first the papillæ are flattened or obliterated; next the pyramids, and finally the cortex are gradually annihilated, and the organ is wholly excavated (or sacculated) and transformed into a multilocular pouch filled with pus. Rayer describes and figures examples in which the pelvic membrane was studded with minute vesicles resembling sudamina.

When the disease is due to the lodgement of calculi, ulcerations may exist on the mucous membrane, and sometimes these have been known to lead to perforation, with effusion of pus and urine into the surrounding cellular tissues, or into the intestines or peritoneum. The accidents usually occur after sacculation and dilatation of the kidney have taken place, but sometimes when the organ does not transcend its ordinary dimensions.

When the pus and urine lodged in an excavated kidney fail to find a free exit through the ureter, from blocking up of its channel by a calculus, a clot of blood, thickened pus, tuberculous or cancerous débris, etc., these fluids accumulate behind the obstacle, and distend the organ into an abscess-like cavity (pyonephrosis), which sometimes forms a palpable tumor in the flank. The matter so incarcerated may open a way for itself in any direction—backward through the loin; downward along the psoas muscle into the iliac fossa, or under Poupart's ligament; upwards into the bronchial tubes, though this is rare;

more frequently it penetrates into the duodenum or colon; or lastly, into the peritoneum.

In the cavity of the inflamed pelvis there are often found, in addition to pus and urine, blood, urinary calculi of various shapes and number, calcareous crusts, hydatids, tubercle, cancer, or whatever other foreign or adventitious matter may have been the cause of the inflammation.

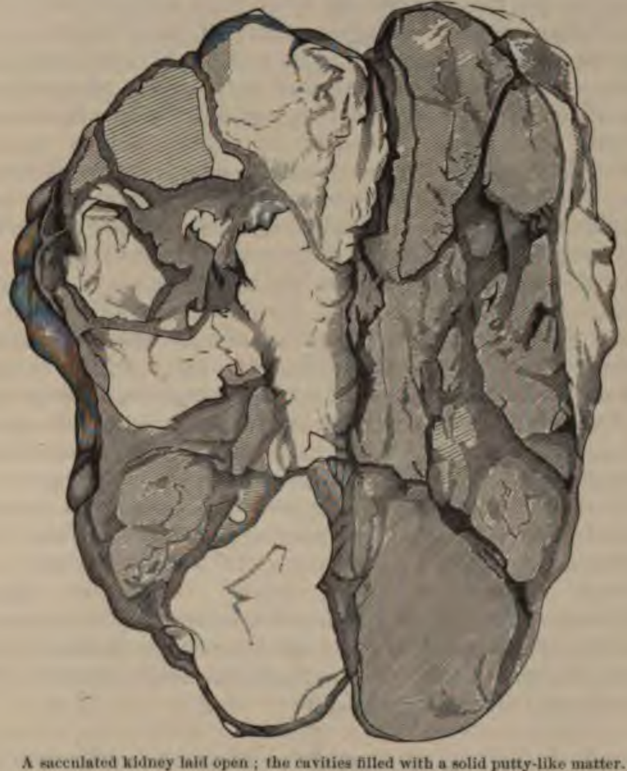
If the urine remain acid, uric acid and the urates may be precipitated in the interior of the pelvis; but if it become ammoniacal, as it commonly does in advanced cases, the mixed phosphates are thrown down. These are sometimes produced in great quantity, and mingling with the purulent contents of the sac, thicken the whole into a semi-fluid mortar-like substance. In other cases, the phosphatic matter forms incrustations, which adhere in places to the walls of the cavity, or lie loose as friable concretions. Sometimes, again, the excavated organ, instead of forming a tumor (or as a subsequent stage to such tumor), slowly contracts, until at length it is reduced to a shrivelled pouch weighing only a few drachms.

In other instances, the pus is gradually inspissated and impregnated with mineral matter (carbonates and phosphates of lime and magnesia (until it is converted into a putty or chalk-like material, which fills up the compartments of the sacculated kidney. Sometimes the fibrous septa which separate the compartments are extensively calcified. In an example of this kind (exhibited by Dr. John Medd to the Manchester Medical Society) which was handed to me for examination, a saw was required to cut the kidney across, and a piece of one of the bony septa which was ground down displayed, under the microscope, the characters of true bone, though in a rudimentary state. A fine specimen of similar transformation is preserved in the museum of the Owens College, of which Fig. 56 is a representation.

It rarely happens, in cases of chronic pyelitis, that the other parts of the urinary apparatus are free from disease. The most common combination is chronic cystitis with dilated, thickened, suppurating ureters. The substance of the kidney, or what remains of it, is likewise involved at length in a degeneration of the nature of chronic Bright's disease (pyelo-nephritis); and if both kidneys are affected, the usual symptoms of that disorder present themselves—general anasarca and characteristic changes in the urine.

ETIOLOGY.—The symptoms of pyelitis, and the varied accidents which it may present during its course, whether acute or chronic, have so direct and intimate a connection with the cause of the inflammation, that practically it is necessary to couple the description of the different species of pyelitis with an etiological condition. This is so far true that the designation pyelitis expresses nothing more than an anatomical fact. As a nosological heading it includes numbers of cases which have little real clinical affinity. In a certain number of cases the inflammation of the pelvis and its appendages is an important, perhaps the most important, feature of the patient's complaint; but in the majority of cases pyelitis is a subordinate and often insignificant incident in the history of some graver disease. Pyelitis may arise under very varied conditions, viz.:

FIG. 56.



A sacculated kidney laid open; the cavities filled with a solid putty-like matter.

(1) From overdoses of turpentine, cantharides, and other stimulating diuretics.

(2) Some degree of pyelitis usually accompanies both acute and chronic Bright's disease and diabetes.

(3) Certain general diseases are sometimes complicated with a degree of pyelitis—typhus and other eruptive fevers, pyæmia, scurvy, diphtheria, cholera, carbuncle, etc.

(4) From mechanical irritation produced by the presence of a foreign body in the pelvis of the kidney or infundibula—urinary gravel and calculi, hydatids and other parasites, blood-clots, cancerous and tubercular deposits.

(5) From stagnation and decomposition of the urine in the pelvis and infundibula. Simple stagnation of the urine (without decomposition), from an obstruction in the ureter, usually causes only dilatation (hydronephrosis); but if it occur suddenly, the pressure of the dammed-up urine may excite acute pyelitis.[1] Severer inflammatory changes occur if the stagnant urine becomes decomposed, and its urea converted into carbonate of ammonia. It is probable that the intractable, generally fatal, pyelitis which sometimes follows pregnancy arises in this way.

(6) From extension upwards of inflammation from the bladder. This is a frequent cause of the worst forms of pyelitis. In whatever manner cystitis may have been engendered — whether by a urinary calculus, enlarged prostate, fungous or tubercular disease of the bladder, or stricture of the urethra—it can scarcely persist in intensity for a lengthened period without producing some or all of the following consequences: dilatation and suppuration of the ureters, pelvis, and infundibula, suppuration extending into the straight tubes and intertubular spaces with formation of scattered abscesses, and ultimately sacculation of the kidneys and destruction of the renal tissue. The term "surgical kidney" has been infelicitously applied to this series of changes by some writers.

(7) From the extension of neighboring inflammation, such as perinephritic abscess, or inflammation connected with caries of the vertebra, as in a case reported by Dr. Cullingworth.[2]

(8) From cold and unknown causes. It is very rare that pyelitis is not secondary to some antecedent morbid process or mechanical irritation; but now and then cases are met with, in which pyelitis exists without any definite antecedent to account for it, as in the following example:

In March, 1857, I admitted into the Manchester Infirmary a man greatly emaciated, with hectic symptoms. The urine contained a large quantity of pus; its reaction was acid; it contained no casts of tubes, nor more albumen than the pus accounted for. The patient stated that his water had been milky for more than a year, and that his health had been gradually failing for about the same time. He had never passed any gravel, nor had he ever suffered from nephritic colic. As far as he knew, the urine had never been bloody. He attributed his complaint to the nature of his occupation, which was to manufacture bichromate of potash. He died eleven days after admission. At the autopsy the thoracic organs were found perfectly healthy, as were also the liver, spleen, and intestinal tract. When the bladder was opened, some injection of the mucous membrane was found, but it was not thickened, and the viscus was not contracted. Both ureters were dilated to about double their usual size, and filled with pus. The two pelves and the infundibula were enlarged, and their lining membrane opaque, and bathed in pus. The kidneys presented very slight signs of disease; the papillæ were flattened and yellowish, as if they contained pus within their ducts; the remainder of the renal tissue appeared healthy. No foreign body was detected in either pelvis, and the path of the urine was free throughout. Death could only be attributed to the long-continued exhausting purulent discharge, which had been allowed to go on without an attempt to check it until within eleven days of his death.

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[1] *See* a case reported by Brunner in the Verhandl. d. phys.-med. Gesellsch. in Würzb., viii. p. 146.

[2] Lancet, 1880, i. p. 14.

SYMPTOMS.—The symptoms of pyelitis are compounded of those directly due to the inflamed state of the pelvis and calices, and of those of the primary lesion which is the exciting cause of the inflammation. Only the former will be dealt with in this connection; the latter will be described under their appropriate headings.

An aching pain and sense of weakness in the back are rarely altogether absent in pyelitis. This pain may be confined to one loin or affect both, according as the disease is single or double. Sometimes, however, single pyelitis is accompanied with pain over both kidneys. The pain is increased on pressure.

Symptoms of nephritic colic are generally noted at one time or other, or repeatedly, when the disease is due to the lodgement of a stone. Similar attacks are also common in pyelitis from hydatids; sometimes also in tuberculous and cancerous pyelitis.

The most important *direct* symptoms of pyelitis are found in the altered characters of the urine. In the early stage the urine contains blood (often only in microscopic quantity), mucus, and epithelial cells from the pelvis and infundibula. The appearance of these last affords the most certain diagnostic indications. The pelvic and infundibular cells are very irregular, spindle-shaped, tailed, three-cornered, elongated, rudely circular, etc. (*see* Figs. 23 and 53). The urine is usually acid. The quantity of albumen in it only corresponds to the admixed blood and pus.

In the more advanced stages, the characteristic epithelium just referred to is usually replaced by pus, which may be discharged in large quantities. The urine is still commonly acid; but as the sacculation of the kidneys proceeds, the mingled pus and urine are liable to decomposition, and the urine becomes ammoniacal.

If the urinary channels remain free, the discharge of pus is constant and regular; but if, as frequently happens, the ureter is blocked up by a calculus, a hydatid, a clot of blood, a mass of viscid pus, or other débris, the discharge of pus is for a while arrested; and if the disease be confined to one side, the urine temporarily recovers its transparency and healthy characters. When the obstacle gives way, pus suddenly reappears in great quantity in the urine. If the distention of the pelvis have proceeded to the formation of a tumor in the flank, the intumescence is necessarily greatly influenced by the formation and removal of such an obstacle. When the discharge of pus diminishes, the fulness in the flank increases, and becomes painful; when the course of the pus is reëstablished, the tumor suddenly subsides, and the urine becomes again loaded with pus. This train of events throws a strong light on the nature of the case. The stoppage in the ureter may persist for varying periods—a few days or a few months—or it may prove permanent. When both sides are affected the obstruction of one ureter diminishes, but does not entirely dissipate the pus from the urine; the same is also the case when the impediment is partial.

Micturition is always more frequent than natural in pyelitis; and during the nephritic attacks it is painful and incessant.

Rigors are of frequent occurrence, especially when there is tumor; they sometimes assume a quotidian periodicity—recurring every evening with tolerable regularity. Well-marked hectic is often present in the later periods.

The bowels are frequently disordered. Unmanageable diarrhœa usually prevails, induced doubtless by the inflammatory adhesions which take place between the dilated kidney and the colon which passes over it. In other cases (when there is tumor) the bowels are obstinately constipated, and require the frequent use of enemata. This is occasioned by the pressure of the tumor on the colon; in one case, related by Bright, the descending colon was so contracted from the pressure of a pyonephrotic tumor, that it was reduced to the condition and appearance of a thick cord.

The occurrence of *tumor* in the flank is generally a late event in the course of pyelitis. This tumor is usually the seat of fluctuation, often obscure; and is commonly painful, and tender on pressure. It is dull on percussion, except where it is crossed by the colon. When the tumor is on the right side it is separated from the liver by the transverse colon; when, however, adhesions form between the sac and the under surface of the liver, this sign may be wanting. The tumor is subject to important variations of size, as already explained, according to the open or obstructed state of the outflow from it into the bladder. In some cases the tumor is so large that it extends across the middle line; more commonly it amounts only to a fulness in the loin or in the space between the crest of the ilium and the false ribs. The outline of the abdomen is thus rendered unsymmetrical.

The ultimate issues of pyonephrosis are diverse; scarcely any two cases run a parallel course. The various directions in which the sac may burst have already been noticed (p. 453), and the symptoms vary correspondingly. But the sac may not burst at all, and the patient dies exhausted by the wasting discharge. This is indeed by far the most common termination. Or again, things may take a more favorable turn; the discharge gradually diminishes, and, finally, ceases altogether: the sac contracts and dries up, and, if the opposite kidney remain sound, perfect restoration to health takes place. Or the restoration may take place differently: the purulent collection, instead of being discharged, dries up into a putty-like mass, and ceases to give further trouble.

The following abstract of cases will serve to illustrate the course and symptoms of some of the chief types of chronic pyelitis. Other illustrations will be found in the chapters treating of parasites, tubercle, and cancer in the kidneys.

CASE 1. *Double calculous pyelitis* (Dance, "Archives Gén.," xxix. 149).—A young woman, æt. 23, was admitted into the Hôtel Dieu, January 12, 1824. She had experienced, two years and a half before, a tedious illness, which commenced with hæmaturia, accompanied by fixed and continued pains in the renal region. Subsequently, the urine became turbid and purulent; it was passed in small quantity and frequently. At the end of eighteen months, after the application of a large number of leeches to the loins, the health improved. The renal pains gave place to an habitual sense of weight in the loins; the urine, however, continued purulent. Three weeks before, the menstrual discharge was suddenly suppressed from cold; and when the patient came under observation the face was drawn, the eyes sunken; there were severe abdominal pains increased by pressure; these were especially severe in the lumbar region. The urine was turbid, scanty, and voided with pain. Leeches were freely applied. But obstinate vomiting came on, and the patient died in five days.

Autopsy.—The kidneys were enlarged to about a third above their ordinary size; their surfaces were nodulated, and unnaturally hard, but presenting here and there points of fluctuation. On cutting open the organs they were found extensively sacculated and full of pus. The left kidney contained nine calculi, and the right fifteen; these were lodged in the dilated calices. The proper substance of the kidney was expanded and attenuated, but otherwise healthy. The ureters were dark colored, marbled on the surface, and their lining membrane thickened.

CASE 2. *Tumor formed by the left kidney (pyonephrosis), discharging pus copiously both by the urethra and the rectum, depending on a large renal calculus* (Bright, loc. cit., p. 227).—A man, æt. 40, first seen by Dr. Bright, April 30, 1836, had, for the last twenty years, experienced occasional pain in the left side, which he ascribed to a blow; he had likewise, at times, felt pain in passing urine, which was then turbid with deposit; but about three months only before Dr. B.'s visit, had a tumor been detected or suspected in the left lumbar and iliac regions. He was found considerably emaciated. The urine was neutral, with a very disagreeable smell, and contained a large quantity of pus with a little blood. The whole quantity of pus passed daily was from four to six ounces.

A tumor existed on the left side of the abdomen descending far below the umbilicus, hard to the touch, and fixed in the left lumbar and iliac regions. It felt smooth and even, and was rather tender at one point.

A month later (June 1st) the tumor appeared to occupy nearly the situation of an enlarged spleen, but Dr. B. thought he felt the colon passing over it. The urine passed in twenty-four hours contained only three ounces of pus. The perspirations were profuse.

June 6.—He had suffered lately a good deal of pain in the left side, and was evidently feverish; he was accordingly directed to leave off the tonic and nourishing medicines and food he had been taking.

15th.—Two days after the last visit diarrhœa came on, accompanied with tenesmus. The tumor was found now greatly diminished; and on examining the stools it was evident that a large quantity of pus was passing that way. The discharge of pus with the urine was undiminished, but there could be no doubt that the abscess in the kidney had ulcerated into the descending colon. The patient lived for about six weeks after this; hiccup came on and proved very obstinate; and pus continued to be discharged both from the urethra and the rectum.

Autopsy.—When the abdomen was opened, the left kidney was seen occupying the space from the diaphragm to the brim of the pelvis, and along its whole length passed the descending colon, much contracted. There was a small fistulous opening, not larger than sufficient to admit a goosequill, from the sac into the sigmoid flexure of the colon as it passed over the lower part of the kidney just at the point where peculiar tenderness had been early observed, and here the intestine looked a little drawn in. The pus was found to have escaped into the psoas and lumbar muscles very extensively. On removing the left kidney, and examining it more accurately, it was found to contain a large coral-formed, lithic acid calculus, extending its branches into all the cavities of a sacculated pelvis. The kidney was full of pus, and in several parts cerebriform matter was sprouting into the cavities with most luxuriant growth, into which tufts of vessels were seen entering.

Cases of this class being surrounded with more or less obscurity as to the exact state of things within the abdomen, are more instructive when the revelations of the autopsy are at hand to illuminate the clinical history; but the nature of some of the cases which end in recovery is so clearly indicated by their symptoms, that they may be cited with advantage, and without any doubt as to their real nature.

The following is from Dr. Todd (*loc. cit.*, Case 48):

Case 3.—A female, æt. 25, unmarried. She had been passing pus with the urine at least a twelvemonth before admission into hospital, and in considerable and constant quantity. For the last five years she had suffered pain in the loins, referred especially to the region of the right kidney. This pain varied in intensity; it was generally slight and dull, but now and then severe. There had been no symptoms of an acute attack, nor any rigors or vomiting. She never, to her knowledge, voided blood in the urine, nor ever passed any gravel or calculus; nor did she ever seem to have suffered from severe pain in the direction of the ureter.

Rather more than a twelvemonth before her admission into hospital, she was suddenly attacked with retention of urine, which lasted twenty-four hours; and immediately after its cessation, she first began to notice in the urine a sediment, which presented a purulent character. This attack of retention of urine was preceded by slight rigors, but the constitutional disorder was of so mild a character as not to cause her to lie up at all.

When admitted into hospital, pus was passed daily with the urine to the extent of two to four ounces; yet there was but very slight general ailment.

On examination, a very large tumor was found situated in the region of the left kidney, forming a considerable projection beneath the abdominal wall. This tumor, which was three times the ordinary bulk of the kidney, was elastic and yielding to the touch, and communicated the sensation of a soft elastic swelling filled with fluid. There was dulness on percussion all over the surface of the tumor, which was smooth, round, and free from any notches or projections.

The tumor was not tender; the patient could bear it to be handled without pain, unless hard pressure were used, when she complained of a dull pain. Her most urgent symptom was an occasional cutting pain, referred to the neck of the bladder, sometimes accompanied with slight difficulty of micturition. She stated that occasionally she had a sensation of fulness in the left side, which would go off rapidly, as if something had burst, and then there would very soon follow an increased flow of pus in the urine. Sometimes as much as eight ounces of pus would be passed in the twenty-four hours. Dr. Todd diagnosticated a stricture of the ureter, probably near the bladder, causing backward pressure on the kidney, with dilatation and sacculation of the organ.

Eighteen months afterwards, the patient presented herself again to Dr. Todd. She stated that, on quitting the hospital, she went to Brighton; there she improved in health very greatly, and the purulent discharge gradually diminished. On a careful examination of the side there was no trace of tumor; only a few pus globules could be detected in the urine, and it was doubtful whether these were not derived from the vagina the bladder.

Case 4. *Pyonephrosis from calculus in the ureter without purulent urine* (Howison, "Ed. Med. Journ.," 1822, p. 557).—A medical practitioner, æt. 25, had severe and protracted nephritic symptoms on the left side at the age of 15. He recovered from this attack, but during the subsequent years he suffered repeated paroxysms of pain in the left kidney, extending to the umbilicus. The urine was at times scanty, and once or twice slightly tinged with blood, but it never was observed to be milky, or to contain anything like pus.

Between the paroxysms his health was good; he followed the practice of his profession, and underwent a good deal of fatigue. The most distinguishing symptom in these paroxysms was fixed pain, of a gnawing description, extending from the spine toward the umbilical region, increased by pressure, even the slightest, during the severity of the attack. There was habitual constipation, and a most unusual sensitiveness to cold.

His last attack but one occurred in February, 1821; he recovered from this in about six weeks, after being bled to 130 ounces. He became lusty and florid after this attack, and was able to take long journeys in his gig; but riding caused so much pain that he was obliged to give it up.

About September he underwent a good deal of fatigue; and it was supposed that he suffered a good deal of pain, from being observed repeatedly to bend his body forward for relief, although he would not allow it when questioned.

One Thursday evening, towards the end of September, he went to bed earlier than usual, complaining of fatigue. He rose next morning at seven o'clock, and his last fatal attack commenced at eight. The symptoms resembled those of the former paroxysms; there was intense pain in the left renal region, and a hardness was perceived when the hand was applied to the seat of pain, with a peculiar sense of crepitation. The symptoms became rapidly aggravated, and, notwithstanding all the means employed, including the abstraction of blood to the extent of 150 ounces (!), he died on the fourth day.[1]

Autopsy.—The left kidney and pelvis were found converted into a reniform sac, a foot long, and nine inches broad. The surface of this sac was marked out into three lobes. When opened it was found full of a fluid resembling pus, mixed with serum. The renal substance had wholly disappeared, except a few small portions, leaving nothing but a cavernous cyst, consisting of the proper external membrane of the kidney and its internal membrane much thickened. It was divided into three large irregular cells, freely communicating with the dilated pelvis, into the apex of which the ureter (of its natural size) opened. The septa between the cells were hard like cartilage with thickened edges.

The orifice of the ureter was closely blocked up by a small calculus.

CASE 5. *Stone in the bladder for* 16 *years—removal by the recto-vesical operation—death five years after from pyelitis.*—James H., æt. 21, was admitted, under my care, into the Royal Infirmary, September, 1858, laboring under symptoms of stone in the bladder. On sounding, a large concretion was forthwith detected. The patient stated that he had been subject to difficulty and pain in making water since he was five years of age. When he came under treatment he was emaciated almost to a skeleton, and unable to leave his bed. Micturition was excessively frequent, the urine ammoniacal, and loaded with viscid pus. By rest in bed and anodyne treatment, the symptoms diminished greatly in severity; and on the 17th of December he was judged by my colleague, Mr. Southam, who now took charge of the case, to be fit for operation. The stone was removed by the recto-vesical section. It weighed over 4½ ounces, and contained a nucleus of oxalate of lime, overlaid with an immense mass of secondary phosphates.

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[1] It seems highly probable from the narrative that death was directly caused by loss of blood; the patient insisted on venesection; he tightened the ligature, and *bled himself* on the night of his death.

The patient made a slow recovery from the operation. By the end of April, 1859, the fistulous communication between the bladder and rectum appeared closed; and the patient was discharged in excellent health, rapidly gaining weight.

The subsequent history of the case, up to the time of his death, extends over a period of five years. Soon after leaving the Infirmary, the recto-vesical fistula reopened, and it never afterwards could be completely closed. The general health continued good, and no practical inconvenience arose from the fistula, until about a twelvemonth before his death. The purulent discharge with the urine then began to increase; he lost flesh, and gradually sank in the early part of 1864.

Autopsy.—The bladder, ureters, and kidneys were removed entire. The bladder was contracted and thickened; the ureters were dilated to the size of a little finger, and were long and tortuous; both kidneys were extensively sacculated, but not enlarged, and filled with pus; and the secreting tissue was reduced to a thin layer of cortical substance scarcely half an inch thick.

CASE 6. *Pyelitis, with tumor—coming on five months after parturition—improvement.*—B. F., æt. 33, a married woman, who had had nine children, was admitted under my care into the Royal Infirmary, on November 30, 1866. She was suffering from a tumor in the right flank and purulent urine.

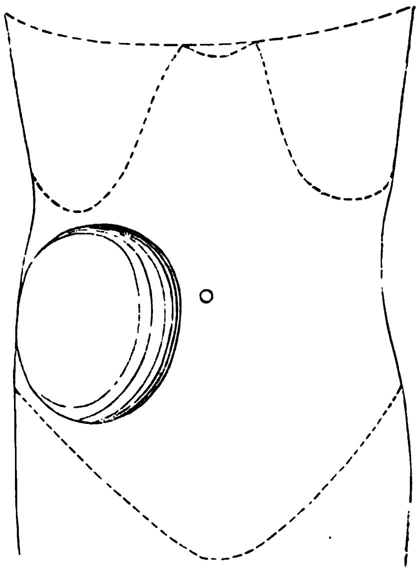
Her last child was born 14 months ago, and five months afterwards she was suddenly seized, while scouring the floor, with stabbing pain in the right side, just under the costal cartilages. This continued off and on for some time, and kept her to bed for days together at times. Somewhere about the same time, she also perceived a small lump in the right flank, which was painful on pressure. About three months later she noticed a white discharge in the urine, and this has continued ever since. She has never had rigors nor pain in micturition. The bowels have been irregular, diarrhœa alternating with constipation.

State on Admission.—The patient was considerably emaciated, with a dirty sallow skin and complexion. The lungs, heart, and liver presented no signs of disease. The abdomen was flaccid and somewhat prominent. In the right loin a smooth, elastic, globular tumor was felt which was not distinctly fluctuating. The tumor was about as large as a child's head; it occupied the entire right flank, and extended inwards almost to the umbilicus and downwards into the right hypochondrium. It was perfectly immovable, and very tender on manipulation. The flank was dull on percussion, but the front of the tumor was traversed by the ascending colon, which could sometimes be distinctly traced over its surface either by palpation or percussion. Between the tumor and the hepatic limits a line of clear percussion could be traced (*see* Fig. 57).

The urine was acid, specific gravity 1020, turbid, with a thick deposit of pus at the bottom of the glass. It only contained albumen to a degree corresponding with the quantity of pus.

The patient remained in the Infirmary for three weeks. About three ounces of pus were discharged daily with the urine. In the second week of her stay, a febrile exacerbation took place, the tumor became more painful, and the temperature rose to 101° in the morning—its usual average being about 99°. The pyrexia subsided in three days, and this was coincident with the discharge of a large quantity of pus. One day she voided as much as eight ounces of pus with the urine. The tumor was considerably reduced in size, and became more flaccid after this, and the appetite and strength improved. The reaction of the urine was throughout acid, though often only faintly so. She was discharged, at her own request, on December 23d.

Fig. 57.



Case of B. F. Diagram showing the position of the tumor.

Though the correctness of the diagnosis was not, in this case, verified by a *post-mortem* examination, it scarcely admitted of any doubt. The acid reaction of the urine, and the correspondence in the size and tension of the tumor with the removal of pus discharged in the urine, together with the physical signs, clearly indicated the existence of a pyelitic tumor; and it is probable that the origin of it was connected with old pyelitis coming on during the patient's last pregnancy.

Case 7. *Pyelitis with tumor, after parturition* (Bright's "Memoirs on Abdominal Tumors, New Syd. Soc." p. 212).—A woman, æt. 30, was admitted into Guy's Hospital, June 13, 1832. She had a large abdominal tumor. It occupied a situation which extended over nearly half the abdomen, not very different from that of a greatly enlarged spleen, but running back more completely into the lumbar region, and there affording a tense, somewhat elastic feel. It appeared to be perfectly fixed; even when the patient was turned completely on the right side, it did not shift its place. It felt as if fixed to the ribs themselves, under their margins, which were obviously protruded a little by its bulk. Towards the lower parts, and particularly below the crest of the ilium, and descending towards the pelvis, the enlargement felt much softer and less tense. Dr. B. was at once convinced that the tumor depended on a diseased kidney, and it seemed likely that the softness of the lower part might arise from a portion of the intestine, which probably was the colon passing over the kidney.

Three years before, the patient had suffered for many months from frequent micturition, with pain and forcing—the urine being occasionally tinged with blood. Eighteen months after, she was put to bed with a living child, and about six weeks subsequently, she first discovered the tumor. Since that, however (nine months before her admission), she had borne another living child, and about Christmas she began to pass considerable quantities of what she considered "matter" with the urine.

On admission she was feeble, and looking hectic, with frequent calls to pass urine, and pain in doing so. The urine, which was acid, contained pus. Some days the quantity of pus was very small; but on other days as much as six or eight ounces of pure pus were collected; and after a large discharge, the tumor was often decidedly reduced for a day or two. The bowels were costive. About the 13th of July, chest symptoms set in, with diarrhœa, under which she sank.

Autopsy.—The tumor proved to be the distended left kidney reaching from the diaphragm to the brim of the pelvis. The descending colon, contracted like a thick cord, ran longitudinally on the surface of the tumor. The tumor was adherent to the colon and the lumbar parietes. The flattened pancreas lay across its surface, on its anterior and inner aspect. The ureter was thickened, and resembled an artery, but its canal was by no means proportionably large. It was traced to the bladder, where its orifice formed a permanent opening, into which a goosequill could easily have been inserted, and the membrane was tuberculated. The bladder was exceedingly small; the uterus natural. The tumor contained about a pint and a half of healthy, well-formed pus, lodged in cells communicating with the pelvis of the kidney, and apparently formed by the distended infundibula.

The right kidney was healthy, as were also the other abdominal organs.

DIAGNOSIS.—(*a*) *Pyelitis without Tumor.*—In the first stage of the complaint, the presence of the characteristic epithelium of the pelvis and calices in the urinary deposit, generally suffices to indicate the nature of the disease. When the urine has become purulent, these may still be found mixed with the pus corpuscles; but in more advanced cases this valuable sign is no longer available, and the source of the discharged pus must be traced by other indications. . These indications are often more of a negative than positive character. When pus is discharged with an acid urine, and signs of disease of the bladder, prostate, and urethra are absent, the *primà facie* inference is, that it comes from the pelvis of the kidney:[1] this interference is strengthened almost to a certainty, if tenderness exist in either loin, or if there be any history of antecedent nephritic colic.

It is much easier to recognize the existence of pyelitis when it stands alone than when it coexists with, and perhaps is the consequence of, chronic disease of the lower urinary passages. Pyelitis is a common complication of old-standing cases of cystitis, enlarged prostate, and urethral stricture. In the absence of tumor in the flank it may be impossible, in such cases, to arrive at a positive certainty as to the coexistence of pyelitis. Little help can be obtained from the character of the urine, because it bears the stronger impress of the vesical, prostatic, or urethral disorder: but a careful weighing of the following points will generally lead to a correct conclusion. The upper urinary passages are likely to be involved when the quantity of pus is very great—two or three ounces or more per day; when, with a large discharge of pus, the urine is only feebly ammoniacal; when the loins are painful on pressure; and the febrile movement and the decay of strength seem out of proportion to the vesical or urethral mischief; lastly, when the latter has been in existence for several years.

(*b*) *Pyelitis with Tumor—Pyonephrosis.*—Cases of this class do not usually present much diagnostic difficulty. There is an elastic fluctuating enlargement on one side of the abdomen, occupying the situation of a renal tumor, and a great discharge of pus with the urine. This discharge is apt to vary from time to time; and the dimensions of the tumor are observed to increase and decrease in inverse correspondence.

When the outlet from the sac is permanently sealed the nature of the lumbar tumor is much more obscure. It is liable to be mistaken for hydronephrosis, a hydatid cyst, a perinephritic abscess, or an abscess or cyst of the spleen,[2] or liver. The diagnosis, in such a case, turns first on the existence of a tumor presenting the physical signs of a renal tumor (*see* Diagnosis of Cancer of the Kidney); secondly, on the evidence of fluidity of its contents; and thirdly, on the signs that that fluid is purulent (recurrent rigors and hectic).

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[1] For the diagnostic signs of the sources of pus discharged with the urine, *see* p. 147.

[2] Caffe records a case of pyonephrosis in a Portuguese physician, residing in Paris, which was mistaken for a cyst of the spleen. No pus had ever appeared in the urine; indeed, there were no urinary symptoms at any time. Nélaton punctured the supposed cyst and withdrew 4½ litres of pus. The patient survived 55 days. (Gaz. des Hôp., 1855.)

PROGNOSIS.—The prospects of a patient suffering from pyelitis differ greatly according as one or both sides are affected, and according to the nature of the exciting cause.

Double pyelitis arrived at the purulent stage is a disorder of very grave consequence, whatever may have been its mode of origin, and usually proves fatal in the end. When the disease is confined to one side, the issue may be favorable, either with or without destruction of the kidney. Cases of this last class are not infrequent: numerous examples have been recorded in which one kidney has been found, after death from some other cause, bearing the marks of previous sacculation and suppuration. Sometimes nothing is found in the situation of the kidney beyond the capsule of the gland tightly embracing a urinary calculus; in others, an empty cellular sac; in others, a sacculated pouch completely filled with concrete pus. An example is reported by Kussmaul[1] in which pyonephrosis was encountered (post-mortem) apparently in an early stage of obsolescence. The patient died of constitutional syphilis, with lardaceous liver and spleen, and Bright's degeneration of the left kidney. The right kidney was converted into a soft, thick-walled tumor, as large as a child's head, situated in the right hypochondrium. It was filled with thick, inodorous pus; the renal tissue had totally disappeared. The sac had contracted adhesions to all the surrounding parts. The ureter was adherent to the wall of the sac, so that the escape of the pus was prevented. A probe, however, could be passed along it into the dilated pelvis. The disease was evidently of old date; no symptoms (beyond the physical signs of tumor) referable to it were observed during life; and the cause of its production could not be clearly made out after death.

The gravity of pyelitis has a close connection with the nature of its original cause. Cancerous and tuberculous pyelitis invariably prove fatal: the prognosis is almost equally hopeless when the disease is secondary to enlarged prostate, intractable disease of the bladder, or urethra. The prospect is more favorable, though still exceedingly grave, in cases which follow pregnancy, or depend upon renal gravel, calculus, or hydatids.

When pyelitis is secondary to some acute disease (zymotic fevers, etc.) it is of very slight consequence, and speedily passes away with the subsidence of the primary disorder.

Rupture of the sac into the thoracic or peritoneal cavities is speedily fatal. Rupture into the intestine generally, if not always, proves ultimately fatal; but the sac may open through the loin with a favorable issue; though this is exceptional.

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[1] Würzb. Med. Zeitschr., 1863, p. 43.

TREATMENT.—The chief general indications in the management of cases of pyelitis are: to remove the exciting cause, and to arrest or control the purulent discharge.

When pyelitis is secondary to Bright's disease, diabetes, scurvy, purpura, diphtheria, typhus or other zymotic fever, the gravity of the primary disease so overshadows the secondary affection that the latter rarely demands separate attention. It is only in the rare hemorrhagic examples when the loss of blood by the urine becomes threatening, that the internal administration of astringents and styptics becomes necessary.

The particular treatment applicable to the different species of pyelitis will be found described under the several headings of Concretions in the Kidney, Parasites, Tubercle, Cancer, etc.

The following observations will find their application in those cases, both acute and chronic, in which the inflammation of the pelvis and infundibula is a leading feature of complaint, and the source of the more important symptoms.

If the attack be acute, and accompanied with pain in the renal region, frequent and painful micturition, bloody urine, and fever, the loins should be cupped to eight or twelve ounces; the cupping should be followed up with warm baths and hot poultices to the loins. Warm diluents should be freely administered. Opium and other anodynes are sometimes demanded on account of the intensity of the suffering and evidence of spasm of the ureter.

In chronic cases, when the secretion of pus is profuse, the efforts of the practitioner must be directed to lessen the discharge, and to bring the renal tumor, if there be any, to a state of contraction or of obsolescence, and throughout to keep up the general health to the highest possible standard.

Among the remedies which are available to check the discharge of pus are, the mineral acids, alum, vegetable astringents, tincture of cantharides, balsamic and terebinthine substances. These last are only applicable when the disease is thoroughly chronic, and a stimulant to the mucous membrane is required.

The metallic astringents have also been occasionally employed with success, when other means have failed. Mosler relates the following instance of the good effects of acetate of lead, in a case of uncomplicated pyelitis arising (presumably) from cold:

David G., æt. 19, cutler, came under treatment in August, 1861. In the spring of the year he had been working in a very cold place, and his illness commenced with a smarting pain in passing water. This was followed by the appearance of pus in the urine. When the case came under observation, it was quite uncomplicated; the only complaint was smarting in making water, and a desire to void it about every hour. Compression of the urethra caused no pus to appear at the orifice; the pus was thoroughly mixed with the urine, giving the latter a turbid appearance. After standing, a layer of pus subsided to the bottom of the vessel, about half an inch thick. The microscope brought to view pus corpuscles and various forms of epithelial cells, some of which were fatty. The albumen was no more than corresponded to the amount of pus. The reaction was acid, and continued so throughout. At first vegetable astringents in large doses (10 grains of tannic acid thrice daily) were employed; then balsamic remedies in the form of Griffith's mixture, etc.; then alkalies (sod. bicarb. ʒiij daily). The quantity of pus remained stationary, in spite of all these remedies; but the smarting in passing water had mostly ceased.

In the beginning of October, the patient complained for the first time of pain in the right lumbar region. At that time there were blood-corpuscles in the urine, as well as pus and epithelium. The pus had increased. The alkalies were now combined with the use of warm baths; the blood soon disappeared, and the pains ceased, but the pus continued undiminished.

On the 1st of January, 1862, the use of acetate of lead was commenced in doses of three grains three times a day. At the end of eight days the dose was increased to four grains three times a day. The effect of the treatment on the amount of pus was marked; on the tenth day the quantity was visibly diminished, and shortly afterwards it disappeared altogether. Some months later the patient presented himself again; the urine was found quite free from pus, and the general health blooming. These large doses produced colicky symptoms toward the end of the second week; and there was at the same time a decided, though not great, diminution in the daily quantity of urine.

The tincture of the sesquichloride of iron has sometimes proved of signal service, as in the following example:

B. H., a woman, æt. 51, was admitted under my care into the Royal Infirmary, in December, 1862, in a state of extreme weakness and emaciation. On examining the urine it was found acid, loaded with pus mixed with some blood. Micturition was frequent with smarting pain. Careful and repeated exploration of the bladder failed to detect a stone. The right kidney was painful on pressure, and the anamnesis disclosed obscure history of renal calculus. There was no fulness in the loin. The daily quantity of pus was estimated at three ounces. The deposit in the urine contained no cellular elements except pus and blood.

She was first put on a mild alkaline treatment, with generous diet, and six ounces of wine. No improvement followed; she continued to lose ground, and was unable to leave her bed; the tongue became dry at times, and symptoms of severe hectic showed themselves. The alkalies were then discontinued; and 30 drops of tincture of steel in a wine-glass of water, administered three times a day; the wine was increased to 10 ounces. This treatment was continued for many weeks, and gradual amendment set in. Blood disappeared wholly from the urine, and the discharge of pus was reduced to less than half an ounce. The general health improved proportionally; and in March, 1863, the patient was able to leave the hospital in a fair way of recovery. She afterwards presented herself among my out-patients from time to time for some months, and steadily gained strength. At length she went to her work (weaving), and I heard nothing more of her until April, 1864. All her symptoms had returned in great severity some weeks before. She declined to comply with my recommendation to enter the Infirmary, and, four weeks after, I heard of her death.

Among the general means designed to keep up the vigor of the system, the most important are cod-liver oil, quinine, nourishing diet, and, above all, change of air. Sea-side localities are preferable, and even sea-bathing may be recommended, if the patient's strength permit.

When renal tumor exists, it may be treated like abscesses in other situations, by incision and free drainage, with antiseptic precautions, and in this way a complete cure may be effected. As a rule, however, it is not advisable to take any steps with a view to procure evacuation of the sac through the integuments, unless there be decided indications of pointing. It must be remembered that there is always a chance (supposing the disease to be confined to one side) that, with rest and patience, the pus may become inspissated, and the abscess pass into a permanently obsolescent state; or that gradual emptying of the sac may take place with final atrophy of the renal tissue. The advantages of an expectant treatment are strikingly illustrated in the following case, recorded by Henninger:

The patient received a blow on the left lumbo-renal region in 1848. Obscure, persistent renal pains followed the accident. Three years after, the patient had nephritic colic on the left side, which recurred in periodical paroxysms, resembling ague. In 1852, the attacks recurred about every three days; they were followed by the discharge of a highly purulent urine. Mixed with the pus were found epithelial cells and crystalline deposits. A tense elastic tumor was discovered in the left hypochondrium, extending as far as the vertebral column. After a paroxysm, and discharge of pus with the urine, this tumor was only doubtfully perceptible, but in exploring along the course of the ureter, a body as large as a nutmeg was discovered in the iliac fossa, in the track of the ureter. The nature of the case was now clearly made out to be calculous pyelitis with tumor. The advice of M. Schutzenberger was to establish a fistulous opening in the renal region with a view to provide a safe outlet for the pus, and thus relieve the neuralgic paroxysms. On consulting with Prof. Sedillot, it was agreed to wait the progress of events, in the hope that the renal tissue would be gradually absorbed, and the kidney reduced to a membranous pouch, which, on the cessation of the secretion of urine, might eventually contract. These hopes were realized. A merely palliative treatment was adopted; and six months afterwards the patient saw an end to his sufferings; he has continued since in uninterrupted health.[1]

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[1] Henninger, Thèse de Strasbourg, 1862.

There are cases, indeed, in which the distention of the sac becomes so great, that the peril of rupture into the peritoneum exceeds the risk of making an opening through the integuments.

The radical treatment of unilateral pyonephrosis by removal of the kidney, although several successful cases are reported, must yet be considered as *sub judice*.[1]

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[1] *See* the discussion in the Clinical Society of London. Lancet, 1882, i. p. 527.

CHAPTER VII.

CONCRETIONS IN THE KIDNEYS.

Close examination of sections of the kidney sometimes reveals the existence of numerous yellowish or brownish striæ, running from the papillæ toward the base of the pyramids. These are due to the precipitation of amorphous urates within the straight canals. This is generally only a post-mortem phenomenon: the cooling of the body after death diminishes the solubility of the urates, and causes them to be precipitated in the uriniferous tubes.

In newly born infants who have breathed, such striæ are very frequently found, especially when death occurs between the second and fourteenth days after birth. Virchow is of opinion that such a deposit is due to the excessive excretion of urates due to the increased metabolism of tissue consequent upon the establishment of respiration. In a few cases, however, the deposit has been found in children who have never respired (Hoogeweg and Martin, Ebstein).

A similar precipitation may, however, occur during life, and constitute the first link in a chain of consequences which leads, eventually, to the production of urinary gravel and stone. Uric acid and oxalate of lime may also be deposited in the same manner, and furnish the nuclei of future calculi. Such concretions may be permanently impacted in the uriniferous ducts, and render these impervious, and themselves cease to grow; or they lodge in diverticula or pouches connected with the ducts, and increase in size amid the renal tissue; or, lastly, and most frequently, they are rolled down along the ducts by the stream of urine, and deposited in the infundibula and pelvis of the kidney; and even many thousands of minute calculi, formed in this manner, may be encountered after death, in these situations (*see* case of J. R., p. 412). Agglomerations of larger size may begin in the same way, or the precipitation may first occur in the infundibula and pelvis.

In number, size, and shape, renal concretions present the greatest diversities. A kidney may contain only one concretion, three or four, or several hundreds. In size they vary from a pin's head, or a hemp-seed, to a horse-bean; and if a concretion become permanently lodged in the pelvis or its appendages, it may go on increasing to a weight of several drachms or ounces. Such a calculus is usually moulded to the divisions of the pelvis, and assumes various grotesque, branched, or arboraceous, forms.

The *anatomical changes* produced by renal concretions are, congestion of the kidneys, abscesses, pyelitis, pyonephrosis, and hydronephrosis. These are considered under their respective headings.

SYMPTOMS.—The existence of concretions in the kidney is usually indicated by an aching pain in the loins, occasionally rising into violent paroxysms (nephritic colic). This pain is characterized by its tendency to shoot along the course of the ureters down to the testicles and the inside of the thighs; it is also commonly attended with a sense of faintness, nausea, or even vomiting. The urine, in these cases, is voided with undue frequency, often with pain at the end of the penis, and it is apt to contain blood, pus, and epithelium from the pelvis of the kidney.

The colicky paroxysms are determined by dislodgement of the concretions from one of the infundibula into the cavity of the pelvis, or from one part of the pelvis to another; but the most severe attacks are caused by the passage of it into the ureter.

The descent of a calculus along the ureter into the bladder is productive of very distinctive symptoms. The patient is suddenly seized with intense pain in the region of the affected kidney, accompanied with a deadly faintness, sometimes with cramp and sickness. The pain radiates in various directions, but chiefly along the ureter to the bladder, scrotum, end of the penis, and the inside of the thigh. The testicle is retracted; there is incessant desire to make water, but the flow of urine is either partially or wholly suppressed. In the former case, the urine is high-colored, often mixed with blood, and voided in drops with burning pain. Violent and frequent vomiting follows; the skin is covered with a cold sweat; there is constant restlessness; the patient tosses from side to side, and assumes in succession a score of different positions in the hope of relief. If the symptoms are not speedily relieved, a febrile movement is produced, which, sometimes, attains a high degree, with hot skin, quick pulse, and incessant thirst.

After these symptoms have continued a certain time—it may be hours, it may be days—relief comes, often quite suddenly. The patient feels something drop into the bladder, and, all at once, his agony is past. Sometimes, however, the concretion fails to clear the ureter, and becomes impacted in some part of its course. In this case, the subsidence of the symptoms is more gradual, and less complete. In other, fortunately still rarer, instances the opposite ureter has already been rendered impervious by the impaction of a calculus on some previous occasion, and the blocking up of the hitherto open channel is followed by total anuria, which leads to a rapidly fatal issue. (*See* Suppression of Urine.)

Renal calculi are sometimes wholly latent. They may even attain a large size, and destroy extensive portions of the gland, without betraying their presence by a single symptom. Or, again, renal symptoms may exist for a longer or shorter period, and then wholly and finally cease. This latter event may occur under two circumstances; either the concretion completely occludes the ureter, and determines gradual atrophy of the kidney, or it becomes encysted in a lateral pouch or diverticulum, and ceases to impede the flow of urine and to irritate the mucous membrane.[1]

THE DIAGNOSIS of a calculus, or calculi, in the kidney or pelvis (except in latent cases) is not generally attended with much difficulty. The locality, distribution, and paroxysmal recurrence of the pains, with the pyelitic characters of the urine, are usually sufficient to indicate the cause of suffering. Neuralgia of the lower intercostal and abdominal nerves sometimes presents great severity, and a paroxysmal character. It is distinguished from renal colic by the absence of blood, pus, and transitional epithelium in the urine. More difficult to distinguish are those cases in which nephritic colic is produced by the impaction of blood-clots or hydatids in the ureter; indeed, absolute certainty cannot often be obtained in these cases until the appearance of gravel, hydatids, or clots in the urine sets the question at rest. The antecedents of the patient sometimes throw an important light on the diagnosis, and a knowledge of the nature of a foregoing attack will furnish a key to an existing one.

In the absence of colicky paroxysms—where the symptoms consist only of obscure lumbar pains and slight disturbances of micturition, careful and repeated examination of the urinary deposit becomes the principal means of arriving at a precise diagnosis. If the symptoms be due to calculus, the deposit will, in all probability, contain scattered blood-disks and spindle-shaped, tailed, and irregular epithelial cells from the upper urinary passages. These may be accompanied with pus corpuscles, and minute agglomerations of uric acid, dumb-bells of oxalate of lime, or some other form of calculous deposit (*see* Fig. 58). These unnatural conditions of the urine are intensified by violent exercise, and diminished or altogether suppressed when the patient maintains a state of rest.

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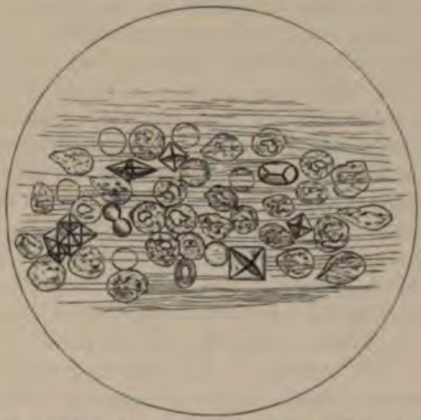
[1] In one case the calculus was discharged externally through a fistulous opening in the loin. (Path Trans., vol. xxvi. p. 128.) In another case, the stone worked its way into the muscles and remained there for seven years, when it was removed by operation. (Amer. Journ. Med. Sciences, April, 1881.)

THE TREATMENT of renal concretions must be modified according to the existing symptoms and the anatomical changes which may be inferred to have taken place in the kidneys.

During the paroxysms of renal colic, the remedies indicated are warm baths, emollient enemata, cupping the loins, and, in highly sthenic cases, venesection. The dolorous spasm of the ureter must be combated by free administration of opium. This drug is freely tolerated in cases of this class, and full doses should be repeated until the system is plainly brought under its influence. When the irritability of the stomach is such as to prevent the absorption of the drug, it should be introduced per rectum or by subcutaneous injection. Belladonna may be substituted where opium disagrees. The secretion of the urine should be encouraged by warm demulcent drinks; hot poultices should be applied to the loins or abdomen, as the local symptoms indicate.

FIG. 58.



Octahedra and dumb-bells of oxalate of lime embedded in a mucoid flake—in the freshly voided urine from a case of renal gravel.

Change in the position of the patient sometimes suffices to dislodge a calculus which lies upon, but has not become fully engaged in, the orifice of the ureter. Manipulation of the abdomen in the course of the ureters may also facilitate the descent of the concretion. Sir James Simpson witnessed relief follow complete inversion of the body.[1]

In the intervals of the nephritic attacks, or when none exist, the treatment must be conducted either with a view to dissolve the concretion (*see* Solvent Treatment of Urinary Calculi) or according to the rules laid down for the management of chronic pyelitis. When abscesses form, or pyo- or hydronephrosis is established, the modes of treatment described under these headings must be followed out.

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[1] Edin. Med. Journ., 1858-9, p. 76.

Incising the kidney through the loins, and extracting the offending calculi through the wound (nephrotomy), is a method of treatment as old as the time of Hippocrates. It is, however, not recommended by modern surgeons, except when suppuration has taken place, and the abscess is manifestly pointing in the loins. When such an abscess is opened, exploration should be made with a probe, and if concretions are detected thereby, cautious endeavors may be made to remove them by suitable instruments. (Hevin and Velpeau—Oldfield, "Thèse de Paris," 1868; *see*, also, "Gaz. Hebd.," 1867, p. 767; "Lancet," 1882, i. p. 184; 1883, i. p. 278.)

Extirpation of the kidney, or nephrectomy, is an operation which has been performed several times in recent years. Eight cases are collected in the "American Journal of Medical Sciences" for January, 1873, p. 277. Six died and two recovered. Another case of recovery after excision of an injured kidney is recorded by Brandt. ("Wien. Med. Woch.," 1873, and "Ed. Med. Journ.," May, 1884.) Dr. Campbell records a case of cystic tumor of the left kidney which was mistaken for an ovarian cyst, and removed, together with the kidney, by operation. The patient slowly recovered. ("Ed. Med. Journ.," July, 1874.) Still more recent successful cases of nephrectomy will be found mentioned in the "Lancet, 1882, i. p. 1070; 1882, ii. p. 568, p. 892; 1883, i. p. 423, p. 424, p. 548, p. 963; while the present position of renal surgery may be gathered from the discussions in the Clinical Society of London reported in the "Lancet," 1882, i. p. 527, and ii. p. 942, and in the Medico-Chirurg. Society; *see* "Med. Times and Gaz.," 1883, i. p. 624.

CHAPTER VIII.

HYDRONEPHROSIS.

WHEN any impediment exists to the flow of urine from the kidneys the secretion accumulates behind the obstruction and distends the parts above. The first effects of the pressure of the accumulated urine are felt in the higher portions of the ureter and the pelvis of the kidney: these parts become dilated. Then the renal substance is compressed, and becomes partially or wholly atrophied and absorbed; so that the organ is at length hollowed out into a pouch or bag, consisting of the fibrous capsule of the kidney. When these changes are associated with suppuration of the lining membrane the condition termed pyonephrosis (already described) is produced. But in a considerable number of instances the obstruction is unaccompanied with purulent formation; the distention proceeds painlessly and gradually. This is the case when the impediment arises from some congenital malformation; also when it is incomplete, or is established by degrees. To this condition the terms "dropsy of the kidney" and "hydrorenal distention" have been applied; but both designations have given place to the term hydronephrosis, introduced by Rayer, and now generally adopted.

MORBID ANATOMY.—Some years ago I exhibited to the Manchester Medical Society a typical example of what may be called a fully developed hydronephrosis. It consisted of a large membranous bag, 13 inches long by 8 inches broad. It represented the right kidney of a woman, who, during life, was supposed to be the subject of ovarian dropsy. She had been twice tapped under that impression, and died of peritonitis after the second operation. It proved, after death, to be the right kidney and pelvis monstrously dilated. When filled with fluid the cyst had a lobed or sacculated exterior, like an enormous colon. The ureter was incorporated with the posterior wall of the cyst, and opened obliquely into the dilated pelvis, with a valvular arrangement resembling that at the entrance of the ureter into the bladder. The channel was pervious to a probe; but the valve-like deformity of its orifice (evidently congenital) prevented the free escape of urine.

On cutting open the cavity a complete fibrous skeleton of the kidney was disclosed (*see* diagram, Fig. 59). The pelvis was dilated to the size of a large cocoanut, and formed a sort of *antrum*, in the interior of which seven smooth rounded openings were situated, large enough to admit the little finger. Each of these openings led into a rudely pyramidal chamber, the bulging base of which corresponded to one of the external lobulations. These chambers were separated from each other by strong membranous septa; but they communicated indirectly with each other through the openings into the enlarged pelvis. Not a particle of kidney substance existed in any part; but three flattened fibro-cartilaginous nodules were found embedded in the outer wall of the sac. The fibrous membrane which composed the pouch and septa was exceedingly tough and strong, much resembling the dura mater. The outer membrane evidently consisted of the thickened and hypertrophied tunica propria, and was continuous with the fibrous structure of the dilated pelvis. The septa corresponded to some of the embryonal divisions of the kidney; and the circular openings represented the chief divisions of the pelvis.

FIG. 59.

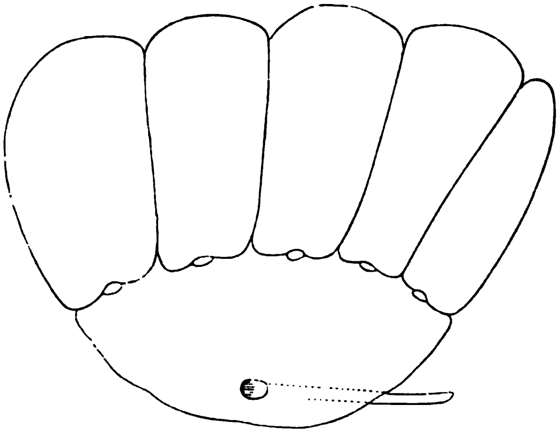

Diagram of a fully developed hydronephrosis.

From this type there are many variations. The sac may not be nearly so large: it may not exceed the dimensions of the healthy organ: it may be even smaller. The chambers vary much in depth, and in number; there may be only two or three; or the whole sac may consist of only a single cavity. The cyst may be composed in varying proportions of expanded pelvis and dilated kidney: sometimes the expansion is almost confined to the former, which is transformed into a globular swelling occupying the hilus of the kidney. The absorption of the secreting tissue is not usually complete. The stagnating urine exerts its pressure in the first instance upon the papillæ which become flattened, and, as it were, effaced; then the bodies of the pyramids are compressed and gradually atrophied; lastly the cortex is encroached on, more and more, until it is reduced to mere islets of reddish tissue on the membranous parietes; and, at length, if life be sufficiently prolonged, these disappear, and not a vestige of the glandular tissue remains.

When only one kidney is involved, a compensating hypertrophy of the opposite organ takes place, and the urinary function goes forward unhindered so long as the latter continues sound, and its channels of excretion free. There is nothing astonishing in this; but it is very unexpected to find that destruction of the secreting tissue may proceed to an extreme degree in *both* kidneys without evoking marked symptoms of deranged urine secretion. A person may apparently exist for a time with the two kidneys wholly reduced to membranous sacs devoid of any tubular structure. In Dr. Strange's case, already cited (p. 232), in which profuse diuresis had existed from infancy, not a particle of renal substance could be detected in the renal sacs after death, though life had been protracted to the age of eighteen years. Another equally remarkable case is related by Faber.[1] The subject of it was a little boy, who had been ventricose from birth, and in weak health. The urine generally presented nothing abnormal; but on two or three occasions the boy suffered from severe paroxysms of strangury, with symptoms resembling those of stone in the bladder. Notwithstanding these drawbacks, the boy was in better health the last year of his life than he had been for the previous four years, and was able to go about. When he had reached the age of 5½ years he fell from a chair and died suddenly in consequence. The autopsy revealed the following state of the urinary organs. Both kidneys were converted into large pouches or sacs, containing no trace of kidney substance. The renal pelves were likewise greatly distended, and the ureters so completely resembled the small intestine that the dissector held them several times in his hand in the belief that they were a coil of intestine. The bladder contained a little turbid urine; its walls were greatly thickened. There was no disease of the prostate, neck of bladder, nor urethra. The entrances from the bladder into the dilated ureters were sufficiently open.

In these and similar cases the atrophy of the secreting tissue had doubtless been going on slowly and progressively, from the time of birth. It cannot be assumed that complete sacculation of the kidneys and total absence of renal tissue existed from birth; for, as was pointed out by Rayer, infants with congenital double hydronephrosis are not viable. Life is probably eked out in such cases by the vicarious activity of the skin and bowels, which undertake some portion of the depurative functions properly belonging to the kidneys. Death commonly takes place, in cases of this class, quite suddenly—sometimes with violent uræmic phenomena.

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[1] Würtz. Correspondenz-Blatt, Bd. xii. 266.

Of 52 cases collected by me, the hydronephrosis was confined to one kidney in 32 instances, and affected both (double hydronephrosis) in 20 cases. When the hydronephrosis was single, the right side was more frequently affected than the left (19 right, and 13 left).

Hydronephrosis sometimes attains enormous dimensions; and fills the abdomen with a soft fluctuating intumescence, reaching from the borders of the ribs to the pubes. Rayer cites an instance in which sixty pounds of fluid were withdrawn from the sac. But the most extraordinary example which I have discovered is the following, related by Mr. Samuel Glass in the Philosophical Transactions for 1747:

> Mary Nix had been remarkable all her life for the preternatural size of her belly. Her mother stated that her daughter was born dropsical; but otherwise she proved healthy; and, notwithstanding the steady increase in the size of the abdomen, she lived to be near 23 years of age.
>
> She is described as a tall and well-proportioned woman, except for the enormous size of her belly; and, for one of so unwieldy a bulk, to have been brisk and active. The menses, which appeared at the usual time of life, continued regular until within eight months of her death. The only complaint was of a pain occasionally felt in making water.
>
> On the suppression of the catamenia, there succeeded a certain amount of dyspnœa, loss of appetite, and emaciation, with swelling of one of the legs, and ulcerations. These symptoms gradually increased until her death.
>
> On taking the dimensions of her body before dissection, the circumference of the abdomen was found to be just six feet four inches, and from the xiphoid cartilage to the os pubis she measured four feet and half an inch! The cutaneous vessels distributed on the abdomen were remarkably large.
>
> The thorax being laid open, the diaphragm was observed to be forcibly protruded into that cavity. The base of the heart lay under the right clavicle, and its apex on the most convex part of the diaphragm; which convexity advanced as high as the third rib. The lungs were surprisingly small, scarcely exceeding in magnitude those of a new-born child. When the abdomen was opened a vast cyst was displayed, from which 30 gallons of a light, coffee-colored, limpid fluid were withdrawn. The fluid was not in the least fetid. In figure, color, thickness, and magnitude, this enormous bag very much resembled the uterus of a cow at the end of gestation. The whole inside was scabrous, and looked as if parboiled, and here and there was observed a small quantity of a coffee-colored sediment. On the left interior part was discovered the orifice

of a duct (ureter) which opened obliquely into the cavity of the sac, and would easily admit a large goosequill. From this opening the tube advanced about twelve inches between the membranes of the bag obliquely upwards, and towards the right, from whence it was deflected downwards and passed between the fold of the broad ligament into the bladder. The abdominal viscera were thrust aside in various directions. The left kidney and ureter were healthy.

The fluid contents of hydronephrotic cysts are generally altered urine. Urea, uric acid, as well as the alkaline and earthy urinary salts, have been found therein. Prout detected urea and uric acid in the contents of a double hydronephrosis from a stillborn infant. Generally speaking, the fluid is much more watery than ordinary urine; and sometimes the organic urinous matters only exist in traces. The fluid may be variously colored; it may contain a little blood, pus, and epithelium, and it is nearly always more or less albuminous.

In the following two cases the contents of the cyst consisted of a substance resembling colloid material. The first is described by Dickinson:

The patient was an old woman of seventy. For twelve years she had perceived a tumor in the left hypochondrium, which at length filled the belly. Constipation alternated with diarrhœa. The patient stated that she occasionally passed "nasty stuff" by the urethra, and that the tumor diminished in size for a time after that occurrence. She at length died of pneumonia. The left kidney was found converted into a large sac about a foot long, divided by septa into compartments. These compartments were filled with a gelatinous substance, which, under the microscope, presented the usual appearances of colloid matter. It lay, however, quite loose in the cyst, altogether unattached to the parietes. There was no obstruction whatever found in the ureter, nor in any part of the urinary channels. Dickinson supposed that an obstruction—probably from a calculus—existed at some previous period, which led to sacculation of the kidney; and that the colloid matter was deposited subsequently. ("Path Soc. Trans.," vol. xiii. p. 137.)

The second case is reported by Prof. Dumreicher, of Vienna, and is remarkable in many ways:

A girl of 13 had observed a swelling in the abdomen from her tenth year. This grew to an enormous size; the circumference of the abdomen, which was uniformly distended, measured 44 inches. The percussion sound was dull, except over a space of four square inches on the left side below the navel. Fluctuation was perceived over the swelling. Prof. Skoda, under whose care the girl first came, diagnosticated an ovarian cyst; but he pointed out the possibility of hydronephrosis. The case then passed to the care of Prof. Dumreicher, who, on account of the dyspnœal distress, punctured through the abdominal wall, and withdrew 18 quarts of a colloidal brown-colored fluid. The circumference of the belly now fell to 30 inches, and the relief to the patient was great. Six weeks later, 16 quarts more were withdrawn; and an injection composed of one ounce of tincture of iodine, in 4 ounces of water, with a drachm of iodide of potassium, was introduced into the cyst. This proceeding proved of no effect. In about a month 14 quarts more were evacuated, and the injection repeated. Severe iodism followed, and continued for a couple of days. The patient then rapidly improved, and left the hospital. In about three months she returned, larger than ever. The belly now measured 46½ inches, and the breathing was much embarrassed; the heart's apex beat in the third interspace. In the course of the succeeding five months the patient was tapped four times, and an aggregate quantity of 37 quarts of fluid was withdrawn—making a total, from the beginning, of 85 quarts! The fluid changed character as the tappings were repeated; it became more and more mixed with blood, and at length with pus. On one occasion a drachm of the crystallized sesquichloride of iron, dissolved in six ounces of water, was injected. This was followed by severe symptoms. At the last, a fistulous passage into the cyst was kept open by an elastic catheter, through which the cyst was evacuated twice daily, and washed out with warm water. Notwithstanding these precautions, the contents of the cyst grew daily more foul, and the patient's strength steadily diminished. She died after having been under observation about a year. On opening the belly the cyst was found to be the right kidney enormously dilated. The sac was intimately adherent to the liver; and the right lobe of the latter was so compressed that it was reduced to half the size of the left lobe. The cæcum and the end of the ileum were fixed by adhesions to the front of the cyst; the rest of the bowels were thrust into the left hypochondrium. When opened, the sac was found in some places thin, in others several lines thick; it was divided into compartments, of which the parietes were traversed by broad membranous bands in various directions, which divided the cavities into a number of small loculi. In these latter a number of cysts with yellowish contents were situated. The anatomical cause of the distention was not very clearly made out; but it appeared to consist in a congenital obliquity of the origin of the ureter, whereby a valvular condition was induced, which impeded the flow of urine. The ureter, after its origin in the cyst, ran in a half circle, downwards and backwards, intimately adherent to the cyst walls and compressed by them. A small supernumerary renal artery arose from the aorta a few lines below the principal branch. The left kidney was enlarged, but healthy. ("Wiener Med. Halle," 1864, p. 139.)

Etiology.—The anatomical conditions which lay the foundations of hydronephrotic distention of the kidney are exceedingly varied. Out of 52 cases which were collated for the purpose of the present article, there existed congenital malformation in 20 cases—affecting the kidney, the ureter, or the renal artery. In two of these, a supernumerary renal artery crossed and compressed the ureter near its origin; in four, the ureter was congenitally imperforate; in three, the ureter entered obliquely into the pelvis of the kidney, creating a valve-like impediment, which necessarily increased as the pelvis expanded. In a case of double hydronephrosis, observed by myself, the details of which follow, the left ureter was greatly narrowed at its origin, and passed obliquely into the dilated pelvis; while the right ureter, which was perfectly normal in calibre, was compressed at its point of exit from the pelvis by an irregular branch of the renal artery. In a case recorded by Dr. Simpson, the ureters were dilated to the thickness of the small intestine in their entire course, except at intervals where they were folded on themselves, while their vesical orifices were so contracted from thickening of the vesical walls, that they barely admitted the stilette of a blowpipe. In a case recorded by Dr. Hare a very curious deformity was found in both ureters, which he thus describes: "On taking the mass (the dilated kidney) in the hands, and pressing very firmly, no fluid escaped by the ureter; examining into the cause of this, it was found that the ureter, at a little distance from its origin, was coiled on itself—like a turn and a half of a cork-screw brought closely together, and that this coil was adherent to the lower part of the dilated pelvis; above this part, the ureter was slightly dilated; below it, not at all. The coils just mentioned acted as a valve-like obstruction to the course of the urine, for on gently dissecting away, with the point of a scalpel, the tissue which held the coils together and united them to the tumor, the retained fluid rushed readily out by the end of the ureter in a full stream.[1]

In 13 out of the 20 congenital cases, the hydronephrosis was double—that is, it affected both kidneys. Two of these perished stillborn, one lived six hours, one thirty, and one thirty-six hours, while one died twenty days, and another between three and four months after birth: but Dr. Hare's patient (just mentioned) survived to the age of thirty-eight years; and the remaining four lived for periods varying from five and a half to twenty years. We must assume, in these latter cases, that the impediment to the urinary flow was at first incomplete (though the malformation was congenital), and that its effects were not fully developed until a subsequent period, and then probably with extreme slowness.

In an instance cited by Rayer, the obstruction (congenital) was constituted by an imperforate urethra;[2] the bladder ureters, and kidneys were distended into capacious sacs (loc. cit., iii. 504). Phimosis also is given as a cause of hydronephrosis.[1]

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[1] Med. Times and Gaz., 1858, i. 234.

[2] In a case which occurred in the practice of the late Dr. Ritchie, that of a male child, who died thirty-six hours after birth, an imperforate urethra was the obstructing cause; the pelvis of each kidney was dilated so as to admit the tips of two fingers. The kidneys themselves retained the lobulated character of the fœtal organs.

Congenital hydronephrosis is often associated with malformations of other organs—imperforate anus, harelip, clubfoot, etc.

Of the 32 cases in which the obstruction arose later in life, it was due, in eleven instances, to the impaction of a calculus in the ureter; and a similar impediment, although not actually found, was inferred to have existed at some previous period in three others. In five cases, a narrowing or obliteration of the ureter existed near its origin or its termination, produced presumably by some past inflammatory or ulcerative process, followed by subsequent constriction. In three cases reported by Dr. Simpson, the ureter, which was of normal, or greater than normal calibre, was compressed immediately above the pelvic brim by a thickened tendinous band of the peritoneum, apparently the result of old inflammatory action. In six instances, the ureters were compressed near their entrance into the bladder by a pelvic tumor—gravid uterus, ovarian cyst, or a cancerous growth: cases of this class are no doubt much more frequent than these numbers indicate; but they are generally slight in degree, and seldom go on to the production of a palpable tumor in the flank.[2]

In a number of the cases collated, a mechanical cause for the distention could not be assigned, or such a cause was only obscurely indicated. In some of these, no doubt, a more careful inquiry would have solved the difficulty; but still there are cases which must at present be regarded as mechanically inexplicable.

In a few cases hydronephrosis has followed upon an injury to the region of the kidney.[3] It is probable that in such cases the injury has been followed by inflammation and consequent formation of cicatricial tissue which has compressed the ureter.

The two following cases illustrate in a striking manner how a congenital malformation, which, at first, only offered a slight obstruction to the course of the urine, comes, step by step, to constitute a greater obstruction, and at length produces fatal results:

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[1] *See* a case reported by Dr. James in the Edinburgh Medical Journal, 1877, p. 135. He believes that the frequent micturition was the proximate cause, the contractions of the bladder preventing the flow of urine through the ureters.

[2] Stadfeldt found dilatation of the ureter common in women dying in childbirth, even when there was no lateral displacement of the womb. In sixteen post-mortem examinations he found such a dilatation nine times; it almost always begins where the ureter crosses the common iliac. Hydronephrosis from this cause (puerperal) is much more frequent on the right than the left side. Out of twelve cases, Stadfeldt found it only once on the left. (Monatsschr. f. Geburtsk, 1862, p. 71.)

[3] *See* Croft, Brit. Med. Journ., 1881, i. p. 123; Hicks, New York Med. Record, April 17, 1880, and Solier, Lyon Méd., No. 45, 1880.

T. S., æt. 20, came under my care February 28, 1867. He had been subject, from the age of two years, to attacks of obstruction of the bowels, continuing for four or five days, and recurring at uncertain intervals of a few weeks or months. During these attacks—which of late had been more frequent and more severe—the abdomen became swollen and tender, and there were sickness and vomiting. The condition of the urine had never attracted any attention.

When I was called to see him, he was suffering from one of these attacks. The bowels had not been moved for five days; the abdomen was distended and painful, and there was frequent vomiting, which was not stercoraceous. The urine was reported to be exceedingly scanty.

On the next day (March 1) he passed only four ounces of urine. The characters of this specimen were peculiar. It was mixed with blood, and its specific gravity was only 1008; no renal casts were found, but a large number of transitional epithelial scales, such as line the pelvis of the kidney. The sickness had ceased, but the condition of the abdomen and the constipation remained the same.

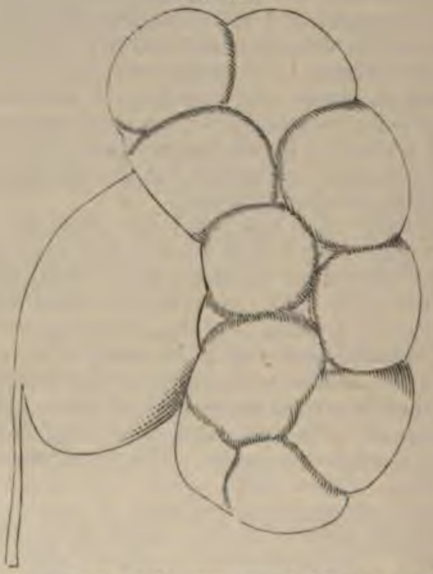
On examining the loins, it was found that there was distinct bulging in both lumbar regions; the bulged portions had an elastic feel and communicated to the fingers an obscure sense of fluctuation. Both loins were dull on percussion, and the dulness reached forward to a line extending from the costal margins to the anterior spines of the crista ilii.

On the next day—seventh day of intestinal obstruction—he passed three ounces of urine, similar in character to that before described. On the eighth day, there was total suppression of urine. On the ninth day, copious discharges of urine took place, amounting in the course of the day and night to more than a gallon. The appearance of the secretion was almost normal. It was straw-colored and clear; it contained no albumen, and only microscopical evidence of blood. It was only unnatural in its specific gravity, which ranged in the different specimens from 1005 to 1007. The bowels still continued without a passage; but a sensible softening of the abdomen had taken place, and the elastic swelling on the left side was very decidedly diminished in size. On the tenth day the urine flowed freely, and fully a gallon was voided before night. It had the same characters as the urine passed the previous day. About midnight, on the tenth day, the intestinal obstruction likewise gave way, and an immense quantity of semi-liquid feces was evacuated. It was now hoped that speedy recovery would—at least for a time—take place; but on the eleventh day the general symptoms were alarming; no urine was secreted, the tongue and teeth became coated with sordes, and the prostration was extreme. On the twelfth day, death took place, preceded by a fit of convulsions, no urine having been discharged for sixty hours.

Autopsy.—On opening the abdomen, two soft, lobulated tumors were found, one in each lumbar region; these were the enlarged and sacculated kidneys. The left kidney was ten inches long by about seven broad—the right about a quarter less. To the bulging inner sides of the left kidney, the descending colon was firmly adherent by a broad attachment for the space of about three inches. It was here that the intestinal obstruction lay; the bowel was contracted at this spot, and tightly stretched over the distended kidney in such a manner as to prevent the free passage of feces. The kidneys, ureters, and renal arteries were carefully dissected out with a view of ascertaining the mechanical cause of the impediment to the flow of the urine, which produced the mischief.

On the left side (Fig. 60), the renal artery was normal in its distribution; but the ureter presented an anomaly. At its origin from the dilated pelvis, the ureter was exceedingly narrowed; its bore was so contracted that only a fine probe could be passed along it. The entrance of the ureter into the pelvis was also oblique, so that a valve-like obstruction was thereby constituted. The action of this latter impediment was clearly shown when the sacculated mass, after being separated from its connection, was held in the hand, and subjected to various degrees of pressure. With moderate pressure no urine escaped from the cut end of the ureter; but when the mass was strongly compressed, the obliquity of the origin of the ureter was for the time effaced, and urine escaped freely. The same thing doubtless happened during life. When the distention of the kidney was moderate, the course of the urine was obstructed; but when the urine accumulated and the distention became great, the obstruction was at length overcome, and the contents of the sac escaped. The lower portion of the left ureter was free from obstruction, and of the usual dimensions. When this kidney was laid open, it

FIG 60.

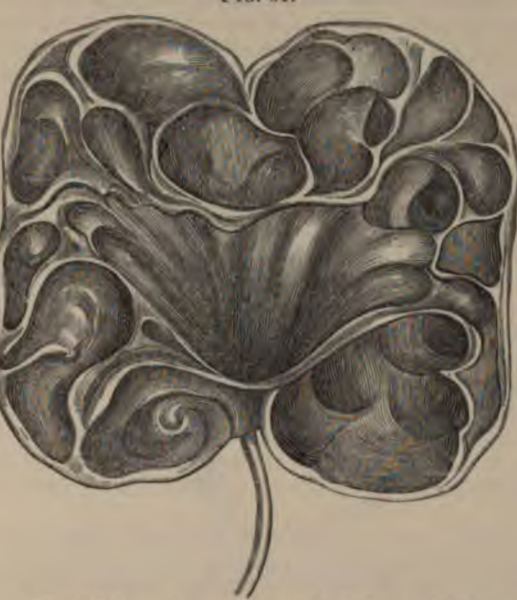


Left kidney, showing the narrowing at the commencement of the ureter and the obliquity of its entrance into the dilated pelvis (about one-fourth the actual size).

(with the pelvis) was seen to be converted into one large lobulated sac, filled with urine (*see* Fig. 61). The renal substance was reduced to a thin layer, varying from a line to two lines in thickness, which formed the outer boundary of the sac. There were no traces of the pyramids. The infundibula and calices were enormously dilated, and constituted the sacculations which gave the mass its lobular character.

The series of changes which brought the left kidney to the state in which it was found, were probably something as follows: The narrowing at the commencement of the ureter was doubtless congenital, and constituted, from birth, a slight impediment to the free escape of urine, and occasioned gradually, in the course of years, by the distending force of the accumulating urine, a dilatation of the pelvis and infundibula, and a progressive excavation of the kidney. As the pelvis became enlarged and distended with urine, it acquired a more globular form, and the orifice of the ureter was, in consequence, carried upwards and assumed an oblique direction, so that an additional obstacle to the escape of urine was thereby created, and one which could only be overcome at intervals, when the pressure from behind became extreme. The adhesion of the colon was doubtless an event of later occurrence, and was the consequence of irritation and inflammation produced by the intermittent pressure against it of the distended kidney and pelvis. It is quite clear that, when this adhesion had once taken place, temporary obstruction of the bowel would arise whenever the kidney became distended with urine beyond a certain point; and that when the increasing

FIG. 61.

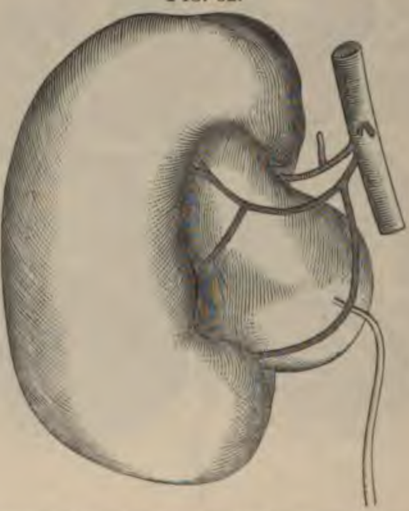


The left kidney cut open (about one-fourth the actual size).

accumulation of urine at length overcame—in the manner already explained—the obstacle to its escape, the renal sac emptied itself, and the intestinal obstruction was also, for the time, removed. In this way may be explained the recurring attacks of constipation and the manner in which relief was accomplished.

The right kidney was in the same sacculated condition as the left, but the destruction had not been carried to so extreme a degree. The mechanical cause of the sacculation on this side was essentially different from that on the left side. There was no narrowing of any part of the right ureter; but at its point of exit from the pelvis it was crossed by an irregular branch of the renal artery (*see* Fig. 62). On this (the right) side, two renal arteries arose from the aorta. The upper artery, after giving off the suprarenal branch, passed into the upper part of the hilus of the kidney. The lower artery divided soon after its origin into two branches, of which one passed into the hilus in the usual way; but the other branch passed downwards to the lower part of the kidney, and, in its course, crossed in front of the ureter just as the latter emerged from the pelvis of the kidney. It is evident that the slight constant pressure of this branch produced a certain degree of impediment to the flow of urine, which in process of time brought about hollowing and sacculation of the kidney.

FIG. 62.



Right kidney (about one-fourth the actual size), showing the abnormal distribution of the renal artery.

The diagnosis was made out in this case with accuracy; and the proposal to puncture the renal sacs was only prevented from being carried out by the large discharges of urine on the ninth and tenth day of the obstruction. Had such punctures been made, however, the relief obtained could only have been temporary: the destruction of renal tissue had gone too far to leave any hope of permanent cure.

A young man of twenty, otherwise in good health, had suffered, from time to time, from paroxysms of pain, followed by nausea and vomiting. On the 3d of February, 1857, he was seized with one of these paroxysms, accompanied with obstinate constipation. The vomiting became intractable; the vomited matters contained blood and sarcinæ; and no passage could be obtained by the bowels. On examining the abdomen, a doubtfully fluctuating swelling was detected in the right flank. The symptoms were attributed to an organic affection of the liver. Under a continuance of these symptoms death took place in five days.

At the autopsy, a bladder-like tumor as large as the fist was found in the right hypochondrium, situated between the liver, the colon, and the duodenum; it was united by adhesions to the two latter. The colon was not constricted at the adherent spot; but the duodenum was so tightly stretched over the tumor that its calibre was almost effaced. The stomach was greatly distended, and filled with a dark-colored fluid.

A closer examination of the tumor revealed the following: It consisted of the pelvis of the right kidney, greatly distended. The right renal artery was abnormally distributed; it divided close to its origin into two branches, one of which ran to the upper, and the other to the lower part of the hilus. The lower branch crossed the ureter near its origin, and exercised a certain compression upon it. The enlarged pelvis pressed forward between the two branches of the renal artery, in such a manner that the origin of the ureter was drawn beyond the level of the lower renal artery, compelling the ureter to loop itself round this branch in order to reach the bladder. Thereto was added a third mechanical obstacle, namely, the adhesion of the ureter in the first part of its course to the outer surface of the distended pelvis, for the space of three-quarters of an inch.

The enlarged pelvis contained ammoniacal urine, mixed with blood and mucus. The corresponding kidney was long and narrow, but otherwise healthy, and scarcely atrophic. The left kidney was natural. (Boogard, "Arch. f. d. Holländische Beitr. z. Natur und Heilk.," Bd. i. p. 196.)

The explanation of these appearances seemed to be this: First, the lower renal artery compressed the ureter, and prevented the pelvis of the kidney from properly emptying itself until a certain pressure was exerted on its walls by the accumulated urine. This impediment was intensified by the curving of the ureter round the lower renal artery. The pressure so exercised probably excited inflammation and adhesion of the ureter to the outside of the expanded pelvis, and again of the latter to the colon and duodenum. The symptoms during life were thus explained. The periodical attacks of nausea and vomiting depended on the periodical dilatation of the sac and the pressure of it on the duodenum. Evacuation of the sac, when the pressure of the accumulated urine reached a sufficient height to overcome the obstructions, caused the paroxysms to subside. In the last paroxysm the resistance proved more obstinate; the duodenum became altogether occluded—hence the constipation; and the portal vessels became probably implicated—determining effusion of blood into the stomach, and hæmatemesis.

Hydronephrosis arises under such a variety of anatomical conditions, that its general etiological relations offer, as might have been expected, little that is characteristic. No age is exempt—not even fœtal life; nor is any especially liable: the two sexes, in the cases collated by me, were found nearly equally represented—25 were males and 23 females; in 4 infants the sex is not mentioned.

THE SYMPTOMS of hydronephrosis depend mainly on the nature of its anatomical cause and on the size of the sac. If the sac be small and the opposite kidney sound, symptoms may be altogether wanting; old age may be reached without suspicion that one of the kidneys has been changed into a membranous sac, and the anomaly may be first discovered at the autopsy.

Generally, however, the distention goes on to the formation of a palpable tumor in the abdomen; and sometimes, as we have seen, this tumor attains an enormous size. Setting aside the cases which perished stillborn, or within a few weeks of birth, there existed among the 42 remaining instances 25 in which abdominal intumescence was detected during life; in 19 of these the tumor was confined to one side, in 6 a double tumor existed.

In its topographical character a hydronephrotic tumor presents the general physical signs of renal tumor. The swelling is situated in the flank; it reaches backwards in the lumbar region to the spine, upwards into the hypochondrium, downwards into the iliac region, and forwards to the umbilicus—encroaching on these regions variously according to its magnitude. The colon is usually in front of it; and the small intestines are thrust into the opposite side of the abdomen. Of the several displacements of the organs on either side I need not add anything to what is detailed in the chapter on cancer of the kidney, where the general characters of renal tumor are fully described. The special characteristics of hydronephrosis are its soft undulating feel; an outline, which is sometimes distinctly lobulated; and the evidence of fluctuation. There is one peculiarity which is pathognomonic when present, namely, the sudden diminution or disappearance of the swelling coincidently with the sudden discharge of a large quantity of urine. This sign is not always available; but it is sufficiently frequently met with to give it an important diagnostic value. It occurred in 9 out of the 25 cases in which the existence of a tumor was clinically ascertained.

In Dr. Hare's case of double hydronephrosis, already alluded to, subsidence of the tumor on the right side took place, from this cause, on several occasions the tumor each time reappearing: that on the left side also disappeared but did not return. Such cases of "intermittent hydronephrosis" are very rare. Another case of the kind reported by Mr. Thompson will be found below (*see* page 497). Mr. Henry Morris has collected six cases in which the affection was intermittent. To these he has added one case observed by himself, where the intermittent hydronephrosis of the right side was due to a villous growth of the bladder, obstructing the orifice of the ureter, but where the disappearances of the tumor were not associated with any abnormal excretion of fluid.

The tumor is usually quite painless, and unaccompanied by any inconvenience except from its bulk. Occasionally, however, tenderness exists over it; and the action of the bowels is irregular. When the dilatation arises from the impaction of a calculus, symptoms of nephritic colic occur at the time when the impaction takes place; or from time to time thereafter, if, as is most usual, some quantity of urine still continues to trickle past the calculus. Similar paroxysms are recorded in two instances where no calculus existed.

The state of the urine usually furnishes no information: in the great majority of cases it is natural; sometimes, however, it contains a little pus; but never in quantity. During the attacks of nephritic colic it may contain blood; and be discharged with great pain, retraction of the testicle, vomiting, etc. The history of these attacks sometimes yields an important clew to the nature of the case. When both kidneys are affected, symptoms indicating defective elimination of urine (uræmia) necessarily show themselves at length. A hydronephrosis implicating one kidney only may, as we have seen, cause little or no inconvenience for many years, even though its bulk be considerable. The opposite kidney performs a double duty and becomes correspondingly enlarged. An individual in this condition, however, leads an existence of considerable peril: for if anything happen to impede the function of the single kidney on which life depends, dangerous symptoms necessarily arise. Rayer supplies the following instructive example:

M. V., æt. 64, had experienced, at the age of 22, pain in the right renal region, shooting obliquely towards the bladder in the direction of the ureter. This pain proved obstinate, and increased more and more; the urine was occasionally bloody, and sometimes of a dark color; the patient became pale and thin. Little by little the urine ceased to contain blood, and reassumed its normal characters; the general condition was perfectly restored; and for a long series of years M. V. enjoyed blooming health.

About the year 1820, M. V. began to grow stout; the belly became remarkably large; and latterly his great size considerably impeded progression.

On the 18th of September, 1834, M. V. experienced an uneasiness in the abdomen which constrained him to keep his bed; pains were felt all over the abdomen, but especially towards the region of the left kidney. This region was tender on pressure; the patient passed no urine; and the bladder was not distended. During ten days M. V. had no desire to void urine, and at the end of this period he only passed two glasses of a citrine color. On examining the abdomen a voluminous tumor was detected, extending obliquely from the right hypochondrium to the left iliac fossa. Obscure fluctuation was felt in the tumor, which was considered to be formed by the distended right kidney (this was confirmed at the autopsy). The condition grew more and more serious as the suppression of urine continued—the tongue became covered with a slimy coating; the features altered; the nights were sleepless; the pulse failed; hiccough supervened; and the patient expired on the 13th of October, 1834.

On opening the body the right kidney was found prodigiously distended, and converted into a pouch filled with 7 lbs. 11 ozs. of a viscid fluid; the tumor was 16 inches long from above downwards, and 7½ inches broad. The ureter was dilated at its origin, but soon underwent a sudden constriction; in this strangulated part a little calculus could be felt which had completely obstructed the duct. Below this obstacle, the ureter resumed its ordinary dimensions. The left kidney was considerably tumefied and reddened. The pelvis was notably dilated and covered with vascular ramifications; the left ureter, like the right, contained a small calculus lodged five inches below the pelvis. The bladder and other abdominal organs were healthy. The state of the right kidney explained perfectly the former ailments of M. V., and death was the consequence of the disabling of the solitary kidney on which his life had so long depended. ("Mal. des Reins," t. iii. p. 490.)

TERMINATIONS.—Hydronephrosis may terminate in various ways. The obstacle may be dislodged, and the contents of the sac discharged, without subsequent reaccumulation. If, in such a case, a portion of the renal tissue be preserved, the organ will be enabled, in part, to resume its function. If the distention has been long established, and the secreting tissue extensively or totally absorbed, the organ after evacuation of the fluid shrivels up into an empty sac. These may be regarded as the most favorable modes of termination.

I find that out of 40 fatal cases, which supply information as to the cause of death, 16 perished from some other disease. Nearly all of these were slight, unilateral cases, which were latent during life. Five cases of double hydronephrosis perished stillborn or soon after birth from abeyance of the urinary function. In 19 cases death took place at a later age as a direct or indirect consequence of the renal distention. Of these 19, one died wearied out with the bulk of the tumor and dysenteric diarrhœa caused thereby. Mr. Glass's patient died from pressure of the vast sac on the respiratory organs. Five cases, in which double hydronephrosis was established gradually, died from progressive abolition of the renal function—four of them with distinct uræmic symptoms, one of whom (*see* p. 487) had also intestinal obstruction due to constriction of the descending colon, which had become firmly adherent to the hydronephrotic kidney. Two more, with single hydronephrosis, died from suppression of urine through impaction of a calculus in the opposite ureter. In four cases, repeated tapping was followed by suppuration of the sac and exhausting hectic: in one, Dr. Hiller's case, the patient succumbed to acute tuberculosis. In another case, the second tapping was succeeded by fatal peritonitis. Pressure of the tumor on the adherent duodenum, and consequent intestinal obstruction, caused death in one instance (p. 491). It is remarkable that only in one solitary instance (to be presently cited) was death caused by spontaneous rupture of the sac.

DIAGNOSIS.—The diagnosis of hydronephrosis is certain and easy only when subsidence of the tumor occurs simultaneously with a sudden excessive discharge of urine, or when trustworthy history of such an occurrence can be obtained. When this symptom is absent, the recognition of the disease depends on the ascertainment of the existence of a fluctuating renal tumor, and the absence of the signs of suppuration.

Hydronephrotic tumors have most frequently been confounded with ovarian cysts,[1] ascites, and hydatid cysts. From an ovarian cyst, hydronephrosis is distinguished by the presence of the colon in front of the swelling, and by the absence of a bowel sound on percussion in the corresponding lumbar region. Ascites is distinguished, when the hydronephrosis is single, by the existence of dulness in *both* flanks: but when the renal tumor is double, and both flanks are consequently dull as in ascites, the latter condition is recognized by the change of level assumed by the fluid when the posture of the patient is altered—dulness from dilated kidneys being fixed in its limits however the position of the patient may be changed. A hydatid cyst is generally identified by the escape of hydatid vesicles with the urine, and sometimes by the presence of a hydatid fremitus. In the absence of these symptoms it may be quite impossible to establish the differential diagnosis of these two conditions by physical signs, and inferences must be drawn from the history. It may be of use to remember, that while hydronephrosis is not unfrequently double, a hydatid cyst is scarcely ever so.

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[1] An interesting case of an attempted removal of an immense hydronephrotic kidney, mistaken for an ovarian cyst,—followed by the death of the patient, is reported in the Archiv. f. klin. Chir., 1865. The cause of the hydronephrosis was a valvular flap of membrane at the origin of the ureter, which prevented the free escape of urine. Another case, in which the patient died on the twenty-second day after the operation, is reported in the Berl. klin. Wochenschr., vi. 23, 1869.

Pyonephrosis is distinguished by the purulent character of the urine—actual or historical—also by the existence of more severe constitutional symptoms, and especially of hectic and recurrent rigors. Circumscribed abscess of the kidney, and perinephritic abscess, are distinguished by their more acute course, the presence of pain and the signs of suppuration.

THE PROGNOSIS, although necessarily grave, is less serious than in other kinds of renal tumor. When the affection is unilateral, not only may life be indefinitely prolonged, but there is always a chance that spontaneous evacuation of the sac may take place, or that the cyst may be punctured with success. If the opposite (hitherto sound) kidney show symptoms of deranged function, the gravity of the prognosis is immensely increased. When both kidneys are affected the issue is unavoidably fatal at length; but many years may elapse before the atrophy of the secreting tissue, or the completeness of the obstruction, reaches a degree incompatible with life.

TREATMENT.—If the disease be unilateral, and inferred to depend on the impaction of a calculus in the ureter, precautions should be taken against a similar occurrence taking place on the opposite side. The patient should be directed to keep the urine adequately diluted by systematic potation, especially on going to bed, and to avoid a too highly animalized diet.

In the absence of this indication an attempt may be made to overcome the obstacle, or, if that be impossible, to facilitate the passage of the urine past it. To this end, the tumor should be carefully manipulated or shampooed, from time to time. As the swelling is usually painless, this can be accomplished without difficulty. In a little girl of eight, who came under my care in the Manchester Infirmary, this treatment seemed to be followed by success. She had a soft, obscurely fluctuating tumor on the left side of the abdomen, about the size of a child's head, which was considered to be hydronephrosis. This was diligently manipulated in every direction, with the aid of a lubricating ointment, on alternate mornings. After the third manipulation, she suddenly passed a large quantity of urine, and the tumor forthwith subsided, and did not again return so long as the patient continued under observation.

If evacuation cannot be obtained in this manner, further interference is in my opinion not justified unless the expansion of the sac be such that its pressure threatens serious mischief.

Under these circumstances tapping may be resorted to. The following case, related by Mr. Thompson, of Nottingham, furnishes an example of the successful adoption of this plan; and the reasons set forth by the writer for the selection of the spot chosen for puncture seem to deserve attention. This case is likewise the solitary instance I have discovered, in which death was caused by bursting of the sac into the peritoneum.

The patient came under Mr. Thompson's observation in May, 1851. He was at that time suffering from great pain in the region of the left kidney. There were considerable enlargement and tenderness on pressure extending over the left hypochondriac, lumbar, and iliac regions. Dulness on percussion also existed in these regions. Symptoms of nephritic colic had existed for a considerable period. Similar symptoms had been observed on a previous occasion, which were suddenly relieved after passing, all at one time, more than a chamber-pot full of water, of the color of port wine. On the present occasion, a similar event took place; in about a week the sac had entirely emptied itself through the ureter and bladder. The symptoms disappeared, and the patient apparently soon recovered. In November he began again to suffer from the same symptoms, which increased up to January 27, 1852. At this time the side was greatly enlarged and tender. There was an obscure sense of fluctuation, and the dulness extended to the right as far as the linea alba; backwards to the spine; downwards to the lowest part of the iliac fossa. The organs in the chest were displaced upwards. The patient's sufferings were now so great that it was determined to draw off the fluid with the trocar. There was no doubt that the sac containing the fluid was a dilated kidney, or a cyst connected with the pelvis of that organ; and in either case Mr. Thompson was disposed to select the interval between the last two (floating) ribs near their anterior extremities at which to introduce the trocar. Mr. T. fixed on this spot for the following reasons:

1. Supposing the fluid to be contained in a sac having communication with the pelvis of the kidney, the kidney would lie behind the sac, partly upon the last two ribs, and partly upon the quadratus lumborum muscle, its normal situation upon this side; and if the instrument were introduced at the place indicated, and its point directed a little forward, it would penetrate the sac without any risk of wounding the kidney.
2. If the sac consisted of a dilated kidney, the point selected would still be the best, as it would be near the part at which the organ began to dilate.
3. It would be behind the peritoneum, and therefore there would be less risk of wounding that membrane.
4. If the patient had been tapped in front, the trocar must have passed through the peritoneum twice: first, that portion lining the abdominal muscles, and, second, that in front of the sac; and, supposing no adhesion to have taken place between these two parts, when the instrument was withdrawn, some of the contents of the sac might have escaped into the cavity of the peritoneum and given rise to inflammation. Besides, there would have been more danger of wounding some

of the bowels, should any portion have become adherent by inflammation between the walls of the abdomen and the sac.

The operation was therefore performed between the last two ribs near their extremities. An incision was made through the integuments and muscles; a small exploring trocar was then introduced; and as there was evidence of the existence of fluid, a larger instrument was inserted, with its point directed slightly forward, and eight quarts of dark-colored fluid were drawn off. It was a singular fact (which was explained on examination of the specimen after death), that soon after this fluid was removed, the further contents of the sac flowed in the natural direction along the ureter.

The patient soon recovered; but it was necessary to repeat the operation in December, 1852, when 3½ quarts of fluid were extracted; soon after which, as before, the sac emptied itself through the natural passages.

The patient soon got well, and did not require the operation again until near eight years afterwards (March, 1860). At this time seven quarts were taken away; not long after which the fluid again found its way along the natural passages, and the patient again made a quick recovery, and remained well until September, 1861. When then seen by Mr. T., he was suffering from his old symptoms. On the 5th of October he was suddenly seized with pain in the abdomen, with difficulty of micturition, cold sweats, rapid pulse, and an anxious countenance. He went on pretty well until the 10th of October, when he suddenly became worse and died.

The post-mortem examination revealed intense peritonitis. Three pints of dark-colored fluid (resembling that found in the sac) were removed from the right hypochondriac and epigastric regions. The sac proved to be the distended left kidney—it contained four pints of fluid; a hole was discovered toward the left side anteriorly, where the rupture had taken place. The descending colon lay before and toward the left side of the sac; the ureter entered the cavity of the sac obliquely through the wall of the cyst. This obliquity of the entrance of the ureter offered a probable explanation of the closure of that tube when the sac was full, and the open state of it when the sac was empty. The rupture doubtless took place on the 5th of October, when the peritonitis began; and in all probability there was some escape of fluid, but not much at that time; a further and larger escape took place on the 10th, after which the patient rapidly sank. There was no stone found in the bladder, nor any obstruction in the lower course of the ureter. (J. Thompson, "Path. Trans.," vol. xiii.)

The early history of the case caused Mr. Thompson to surmise that the patient had formerly voided urinary calculi; but none were ever found. It is quite as possible that the obliquity of the entrance of the ureter into the pelvis of the kidney was a congenital malformation, and that this constituted the real cause of the hydronephrosis.

Dr. Hiller relates another case of congenital hydronephrosis repeatedly tapped, in front, with temporary success:

The patient was born with great enlargement of the abdomen, simulating ascites, for which it was mistaken till he was nearly four years old. It was then ascertained to be an enormous cyst springing from the right lumbar region. From its great size it caused difficulty of breathing and prevented his walking. The cyst was tapped in front, and 102 fluid-ounces of clear non-albuminous fluid were drawn off, having all the characters of dilute urine. The fluid rapidly re-collected, and on a second tapping was found to be albuminous and purulent, but still to contain a considerable quantity of urea. Attempts were made to establish a permanent fistula anteriorly, and then posteriorly; but on each occasion the fluid after a time ceased to flow. Much irritation and depression followed the several tappings, so that the patient's life seemed to be endangered. After one of the operations a quantity of fluid was passed from the bladder exactly similar to that from the cyst, and quite unlike what was usually passed from the urethra; a temporary communication thus obviously being established between the cyst and the bladder. When the case was reported, the patient had been left without operation for some months, and had regained his strength; but the cyst remained, varying from time to time in size, and the urine was often purulent and fetid. It was presumed that there was some congenital malformation of the right ureter which rendered it liable to occlusion, but admitted, under some circumstances, of the passage of fluid. ("Brit. Med. Journ.," April 8, 1865.)

The following is an abstract of the completion of this case:

The patient remained under occasional observation between the above date and July 16, 1868, when he was admitted into hospital under Dr. Hillier, suffering from headache and feverishness. During this interval he had been once tapped, and a quantity of urine-like fluid was drawn off. Acute tuberculosis manifested itself, the patient gradually sank, and died on the 5th of August. On post-mortem examination, the great distention of the abdomen was found to be due to the presence of a large cyst, which filled the greater part of the cavity. The cyst, which measured twenty-seven inches in circumference over the long diameter, and twenty-four over the short one, appeared to take the place of the right kidney—the suprarenal capsule, apparently normal, being attached to it. The ureter proceeded from the lower part of the cyst, to which it was attached for about an inch; the orifice of the ureter at its vesical extremity was found smaller than usual. When a small dressing-probe, which was with difficulty passed up the ureter, reached within two inches of the cyst, and was then withdrawn, fluid could be squeezed out of the cyst through the ureter into the bladder *guttatim*. This could not be done previous to the passage of the probe. The cyst contained 83 ounces of clear fluid of a pale lemon color and urinous smell, specific gravity 1002, very slightly acid, with the faintest trace of albumen, and presenting under the microscope a few broken-down cells of large size.

The *left* ureter was about an inch in diameter at its upper two-thirds, natural in size below, but contained calculous matter, forming an obstruction to the flow of fluid, which could, however, be easily overcome. The pelvis of the left kidney was dilated sufficiently to contain a small

pigeon's egg, and contained a little calculous matter, found on examination to consist of uric acid. Tubercles were found in the brain, lungs, liver, and spleen. (" Med.-Chir. Trans.," lii., 1869.)

When the tumor is on the left side, as in Mr. Thompson's case, mentioned above, the best point for tapping, is just anterior to the last intercostal space. Mr. Morris has tested this in the post-mortem room and has found that in no instance was the spleen injured. On the right side, however, such a puncture would pierce the liver, and Mr. Morris here recommends a spot " half way between the last rib and the crest of the ilium, between two inches and two inches and a half behind the anterior superior spine of the ilium."

In recent years the whole mass has been successfully removed by abdominal incision (*see* cases by Dr. Savage and Mr. Knowseley Thornton in the " Lancet," 1880, vol. I.).

CHAPTER IX.

CYSTS AND CYSTIC DEGENERATION OF THE KIDNEYS.

Cysts are found in the kidneys under four practically different circumstances, namely: 1. Scattered cysts in kidneys otherwise healthy. 2. Disseminated cysts in the atrophic form of Bright's disease. 3. Congenital cystic degeneration. 4. General cystic degeneration in adults.

1. Scattered Cysts in Kidneys Otherwise Healthy.—It is not uncommon to find on the surface of healthy kidneys one or more cysts, with delicate walls, varying in size from a pea to a marble or walnut. One or more of similar appearances may also be found in the interior of the gland, chiefly in the cortical substance. Such cysts are filled with a yellowish albuminous fluid—usually diffluent, sometimes gelatinous,—containing phosphates and carbonates, sometimes a large quantity of cholesterine, and very rarely urea and uric acid.

Cysts of these dimensions do not produce any symptoms during life; and their effects on the functions of the gland are insignificant. Sometimes, however, cysts of this class attain a monstrous size, and form a tumor recognizable during life. Mr. Cæsar Hawkins gives an account of a remarkable case in which the right kidney of a boy, six years of age, had an enormous cyst attached to it. The cyst filled the entire right side of the abdomen from the false ribs to Poupart's ligament. The attached kidney was healthy in its structure, and the ureter free. In the wall of the cyst, separated by a distance of five inches from the kidney, there was inserted a small mass about the size of a walnut, which projected into the cavity of the cyst. This body proved to be a third kidney, consisting of a single lobule, with the cortical and tubular part perfect; and having a single mammillary process and calyx; but no excretory duct could be traced. The urine had been natural. The cyst was punctured during life; and about five pints of fluid were found in it after death. The fluid contained neither albumen nor any of the special urinary ingredients.

Dr. Hare ("Path. Soc. Trans.," vol. iv. p. 199) describes a very similar cyst taken from a man aged sixty-two. A tumor was detected during life on the right side, stretching from the ribs to the pubes. After death, a large cyst was found connected with the right kidney. The lower half of the gland was partly spread out over a portion of the tumor, and partly absorbed. The upper half was healthy; nor did it (nor the opposite kidney) contain any other cyst, with the exception of one, about as large as a hempseed. The large cyst contained an almost transparent, pale, yellowish-green fluid, quite limpid and diffluent when the cyst was first opened: but after the fluid had been exposed a few minutes to the air it set into a tremulous jelly.

In neither of these two cases was the pelvis of the kidney nor ureter dilated. In both cases the disease was doubtfully traced to external violence.

2. DISSEMINATED CYSTS IN THE ATROPHIC FORM OF BRIGHT'S KIDNEY.—These have already been noticed in connection with Bright's disease (*see* p. 401.)

3. CONGENITAL CYSTIC DEGENERATION OF THE KIDNEYS.—A considerable number of these curious cases have been published, and most of them have been collated by Virchow in two elaborate papers (Gesammelte Abhandlungen, pp. 837 and 864).

Kidneys in this condition present an enormous proportionate bulk, being as large as, or larger than, the kidneys of adults. In all but two cases both kidneys were affected. In several instances embryotomy was required to effect delivery, on account of the immense size of the abdomen. The fœtus (generally expelled prematurely) is necessarily stillborn if both sides are affected, on account of the pushing up of the diaphragm, and the mechanical obstacle thus created to the expansion of the lungs.

Dr. Lever ("Path. Soc. Trans.," 1848–9, p. 74) has recorded the following typical example. The fœtus was one of eight months. It was clubfooted and clubhanded; it had six fingers on the left hand and as many toes on each foot. There was a hernia cerebri (encephalocele?) at the posterior part of the head. The thoracic and abdominal viscera were natural, except the kidneys. The right kidney weighed 4 oz. 6 drs.; the left, 4 oz. 1 dr.: they were irregular on the surface from numerous projecting cysts. On a section being made through the centre of each, it was found that all traces of kidney structure had disappeared, and that its place was occupied by an infinite quantity of cysts of different sizes, forming the whole mass of the organ; the calices were in part normal, but large, and the pelvis of each kidney was perfect, with the exception that it formed a blind sac, with no opening; that is to say, there were no ureters. The bladder was small and empty; there was no trace of ureters on its external surface; but internally, at the spots where the ureters should have entered, there were small imperforate papillæ.

The structure of these kidneys was examined by Dr. Gull under the microscope. He could not detect any secreting tissue, and considered the cysts to be obstructed and dilated Malpighian capsules.[1]

The degeneration has not always been found in so extreme a degree as in this case of Dr. Lever. Generally, some remnants of secreting texture (uriniferous tubes and Malpighian tufts) have been detected in the interstices between the cysts. In some cases the external surface is smooth, while the interior presents a spongy or cavernous structure, which, under the microscope, resolves itself into myriads of minute cysts. The researches of Virchow and Föster have fully demonstrated, that the cysts in these cases are originally produced by dilatation of short sections of the uriniferous tubes into pouches; these pouches afterwards become enlarged and separated from each other, and at length form distinct cysts. They are lined with a tessellated epithelium, and contain, at first, a urinous fluid, which at a later period, when the cyst attains a larger size, becomes albuminous.

It is curious that malformations of the pelvis of the kidney, of the ureter, bladder, or urethra, or of some other part of the body, nearly always coexist with congenital cystic degeneration of the kidneys. Sometimes, however, the lower urinary passages are perfectly open.

Virchow first pointed out the mechanical cause of this disease. In all the cases examined by him, there was found an imperforate state (atresia) of the straight ducts which terminate on the papillæ; and he conjectures that this had arisen from intra-uterine inflammation of the ducts of the papillæ, which ended in adhesion of their parietes and closure of their calibre. He further believes that the usual cause of this inflammation is the impaction of uric acid or the urates (Harnsäure-infarct), in the straight canals (*see* p. 474). The closure of the excretory ducts necessarily causes stagnation and accumulation of the urine throughout the entire organ, and leads to dilatation of the uriniferous tubes and Malpighian capsules, and the ultimate formation of cysts.[1]

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[1] Dr. Duffey ("Dub. Quart. Journ.," xli. p. 438) has described the following case in an anencephalous fœtus at the full time. The fourth and fifth fingers of both hands and the corresponding toes on the right foot were united by a web up to the second phalanges, and there was a sixth digit similarly united to the entire length of the fifth. The left foot was normal. The abdomen was 18 inches in circumference. There were two tumors found in the abdomen, which proved to be the kidneys, lying on and adherent to which were the ovaries and Fallopian tubes leading to a bifid uterus. The kidneys presented the usual lobulated appearance of the fœtal organs, but on section numerous small transparent cysts were found of the size of peas, in a matrix of a light grayish color, from which they could not be detached, and which contained a clear serous fluid. The distinction between the cortical and medullary portions was totally obliterated, but the outline of the calices could be distinctly traced. The ureters were pervious; bladder empty. The right kidney, even after exposure to the air for some days, weighed 6 oz.; the left was apparently of equal size.

4. GENERAL CYSTIC DEGENERATION OF THE KIDNEYS IN ADULTS. —This is a somewhat rare condition, though most museums contain specimens. There are two very fine examples in the collection of the Owen's College. In this form of disease the organs are more or less enlarged, so as sometimes to weigh several pounds, and to constitute tumors in the abdomen recognizable during life. Both kidneys are always affected; but not, generally, in an equal degree. The substance of the gland is converted into a mass of closely aggregated cysts, lodged in an abundant matrix of connective tissue (*see* Figs. 63 and 64). The cysts do not communicate with each other, nor with the calices —except in rare cases, when some of them suppurate and open into the pelvis. They range in size from a pin's head to an orange, and have walls of varying thickness. Their contents also vary: some contain a limpid yellowish or reddish serum; others a gelatinous substance. The fluid within the cysts always contains albumen, but not urinary ingredients. The interior of the cysts is lined with epithelium usually of the tessellated variety; and sometimes blood-disks, pus-corpuscles, and cholesterine crystals are found within them. In far advanced cases the secreting tissue of the kidney is almost entirely destroyed; more frequently remnants of renal tissue are found in the fibrous matrix between the cysts and in the pyramidal portions. The pelvis, ureter, and bladder are open, and usually healthy. Two or more cysts may become confluent by absorption of some parts of their walls, and then an irregular cavity is produced, with fibrous bands or fræna passing from side to side.

Quekett attributed the formation of these cysts to dilatations of the Malpighian capsules; but the observations of Dr. Conway Evans,[2] and Dr. Bristowe,[3] on what appear to have been incipient cases, lead to the conclusion that they are formed, as in the congenital cases, by expansion of sections of the uriniferous tubes, and occlusion and atrophy of the intermediate portions. Independent sacs are thus constituted, which at first are so minute that they can only be seen with the microscope, but at a later period they enlarge into visible cysts.[1]

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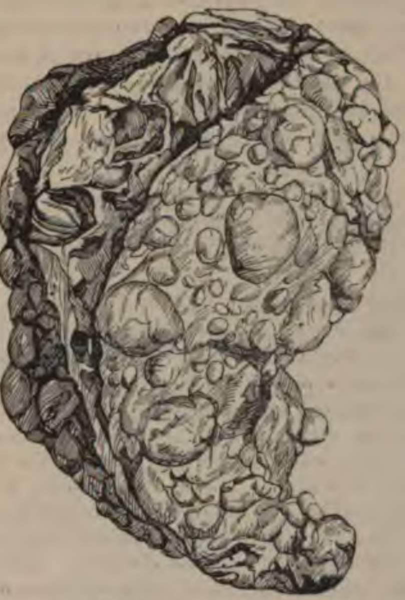
[1] Koster argues that this view is incorrect. He attributes the origin of the cystic kidney to an abnormal development analogous to atresia ani, non-development of the urethra, and similar conditions; and urges in support of this view the total absence of the renal calices and pelvis, and also the ureters in many cases of this disease. Kupfer's investigations into the development of the kidney, showing that the blastema for the tubuli uriniferi is developed by itself, independently of the evolution of the Wolffian duct (which becomes ureter and renal pelvis), will if confirmed tend greatly to strengthen the theory advocated by Koster. Origin of the Congenital Renal Cystoid. Nederlandsch. Arch. v. Gen. in Naturkunde, 1867, translated by W. D. Moore, Dub. Quart. Journ., xlvi. 256.

[2] Path. Trans., vol. v. p. 183.

[3] Ibid., vol. ix. p. 309.

The clinical history of these cases has been but imperfectly studied. Of fifteen cases which I have been able to collect—including one contributed by myself—ten were men and five women; most of them were about the middle age; ten were between forty and fifty years of age; one was "old," one was thirty-nine, and the youngest thirty. The symptoms during life are not very distinctive. The course of the disease is essentially chronic; the secretion of urine goes on to an advanced period, without marked diminution—it may even be greatly increased. An unnaturally low density of the urine would appear to be a tolerably constant feature, at least in advanced cases. The end (if the patient die of the renal affection and not of some complication) is usually sudden, with manifestations of uræmic coma and convulsions. In a case cited by Rayer, there were recurrent

FIG. 63.



General cystic degeneration of the kidney in an adult—from a preparation in the Museum of the Manchester Infirmary—one-fourth the actual size.

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[1] A form of cystic kidney somewhat different from that described in the text is found associated with a similar degeneration of the liver. It must not be confounded with putrefactive changes in these organs. (*See* Brit. Med. Journ., 1884, i. p. 61.)

attacks of excessively violent lumbar pains, severe gastric symptoms, abundant discharge of a watery urine, and lastly, convulsions, delirium, and coma. In another case recorded by the same author, the patient—whose only previous suffering consisted in old-standing dyspeptic symptoms—was suddenly seized with coma, resolution of the members, and convulsive upturning of the eyes, which proved fatal in twelve hours. In two cases reported by Dr. Whipham,[1] the closing symptoms were bronchitis with urgent dyspepsia, without coma or convulsions. Albuminuria and recurrent hæmaturia are among the most constant symptoms. In Dr. Conway Evans's case, the urine was, however, not albuminous on the day of death, nor two months previously. Death was caused in this case by cardiac disease, and the renal degeneration was not, comparatively speaking, very far advanced.[2]

The latent and insidious course of the disease, with sudden stormy termination, is well illustrated in the following example, which occurred in my practice:

On Saturday evening, October 28, 1871, I was requested by Mr. R. Heathcote to see Mrs. S., a married lady of forty-eight. He informed me that the patient was the mother of several children, of whom the youngest was four years old. The menses still continued, but irregularly. Some three months previously she had been under his care for neuralgia and anæmia, which yielded readily to quinine and iron. Since then she had been in her usual fair health up to the previous Monday, when she was suddenly attacked with vomiting. The vomiting continued, almost incessantly, during the current week, and no urine had been voided since Tuesday. Early on Saturday morning, she was seized with an epileptic fit; this was succeeded by two more in the course of the day. At noon, about six ounces of urine were withdrawn by catheter. This was of normal appearance, but it contained a moderate amount of albumen.

When I saw her in the evening, the vomiting still persisted, accompanied with devouring thirst. The patient was nearly fully conscious. There was great restlessness, with tossing of the limbs and moaning.

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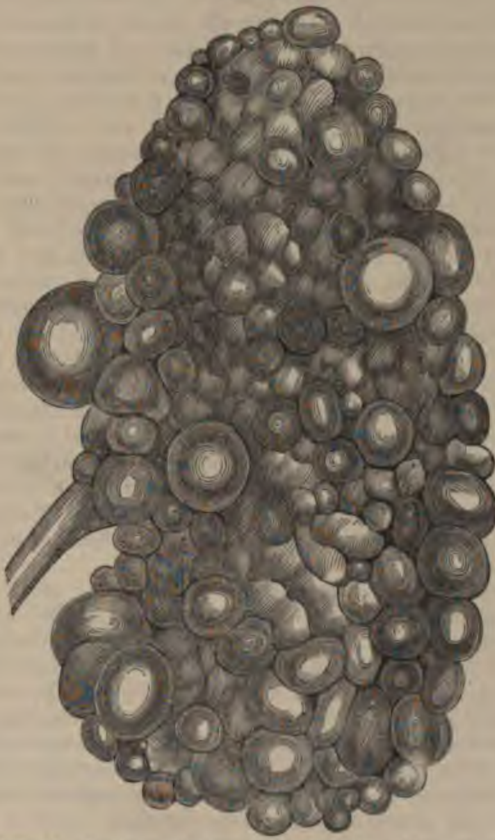
[1] Path. Soc Trans., vol. xxi. p. 244.

[2] [Three cases of cystic kidney have died in the Manchester Royal Infirmary during the last three years. The first case had shown mental depression and stupor for a fortnight before admission, and died a week afterwards with uræmic symptoms. The second case died from uræmic coma two days after admission, and no reliable history could be obtained. Before death the urine was scanty and contained a large quantity of blood. Both kidneys were cystic, and the left had two ureters, proceeding from entirely separate pelves of the kidney; the two divisions of the kidney were equally affected by the cystic transformation. In the third case, the kidney change was found only in the regular course of a post-mortem examination. The patient had died from chronic cerebral meningitis, and during life there was neither albuminuria nor any other symptom of kidney mischief. Microscopic examination showed a certain amount of true kidney structure, but the glomeruli, renal tubes, and arteries were surrounded by a great quantity of fibrous tissue.—R. M.]

The bowels had been opened by injection. The pupils were strongly contracted, and the tongue was dry.

On examining the loins, I detected two soft elongated swellings or tumors, one on each side, in the renal regions. The tumors appeared about the same size, and might be about as large as a cocoanut, but of greater length. They did not fluctuate. Both flanks were dull on percussion. I ventured to express the opinion that we had to do with cystic degeneration of the kidneys, basing the diagnosis on the uræmic complexion of the symptoms, the existence of two soft renal tumors with albuminuria. If this diagnosis were correct, there could be no question of curative treatment. It was suggested, however, that the lumbar swellings might possibly consist of fecal accumulation. The treatment was accordingly directed to remove these. Sulphate of magnesia ene-

FIG. 64.



Left kidney of Mrs S.—showing complete cystic transformation of the organ—about one-half the actual size.

mata were ordered to be repeatedly administered, and belladonna in pill was given by the mouth. Nothing availed. In the course of the night the patient had six epileptic fits, and when we saw her on Sunday morning she was barely conscious. The stomach was, however, much quieter, and she had been able to retain a considerable amount of liquid nourishment. No urine had been voided, but 16 ounces were withdrawn by catheter. This was pale amber, clear, faintly alkaline; it was slightly albuminous (abt. $\frac{1}{12}$), specific gravity 1013. After it had been kept for twelve hours, it deposited a copious sediment of triple and amorphous phosphates, and spheres and dumb-bells of urate of ammonia, but no casts of tubes could be detected. During the course of Sunday and Sunday night, the epileptic seizures recurred again and again, and death took place on Monday morning, after an illness of almost exactly a week.

Autopsy.—The body was tolerably well nourished. Only the abdomen was examined. All the organs in it were healthy except the kidneys. The kidneys presented typical examples of cystic degeneration (*see* Fig. 64). They were considerably enlarged, and appeared to consist entirely of a congeries of large and small cysts. The right weighed 28 ounces, and the left 26 ounces; they were of an elongated, oval form, between eight and nine inches in length, and four inches in thickness. On section of the right kidney, not a particle of normal renal tissue could be seen; the entire organ was converted into cysts and intervening fibrous tissue. The left kidney was not so completely degenerated. The cortical substance was all transformed into cysts and fibrous matrix; but the pyramids were not wholly destroyed. The papillæ could still be distinguished, together with a short length of the annexed portions of the pyramids. These presented a pale-red striated appearance. Judging roughly, about a twentieth part of the renal tissue might be said still to exist, and on the services of this remnant the patient's life must have latterly depended. It is most strange that life could have been protracted until this extreme degree of destruction had been reached.

The cysts varied in size from a pea to a walnut; most of them were as large as marbles. Their contents were (generally) a clear yellow, highly albuminous serum. Some were opaque and semi-solid, either yellowish or dark red. These differences depended on two circumstances, namely, the degree of inspissation and degeneration of the contents, and on effusion of blood into the cysts. Under the microscope, the yellow semi-solid contents were found to be composed of free fatty and albuminous granules and large numbers of so-called "granular corpuscles." A few plates of cholesterine and crystals of triple phosphate were also found. No traces of urea or uric acid could be detected.

The following example from Bright's memoir on abdominal tumors ("New Syd. Soc.'s Publications," vol. vi. p. 208) shows the successive appearance of a renal tumor first on the left, then on the right side of the abdomen, and gives an excellent picture of the disease.

Mr. ——, about thirty, seen by Dr. Bright, November, 1835. His aspect bespoke a man laboring under some formidable chronic disease.

He was evidently much emaciated and greatly enfeebled. He passed a moderate quantity of urine, which was acid, light-colored, and albuminous. His present illness dated about two years back, at which period he had decided hæmaturia, which continued at intervals for some time. Since that, he had never considered himself in health; he had, however, pursued his usual occupation till lately, but for the last four months he had been more decidedly an invalid. A tumor was to be distinctly ascertained in the left lumbar space, where it appeared pretty firmly fixed (*see* Fig. 65). It might be fairly grasped by the hand so placed that the thumb was near the spine, and the finger advanced into the hypochondriac region. The history of the case, the state of the urine, and the situation of the tumor, all led to the easy decision that the tumor depended on enlarged kidney. When felt in front, the spleen, or the descending colon loaded with feces, suggested themselves; but the fact that it seemed to belong rather to the posterior than the anterior part of the abdomen, and its fixed feel would have removed these doubts had not the history of the case pointed so distinctly to the kidney. The exact nature of the renal disease was less obvious. The very considerable enlargement of the organ did not belong to the usual history of albuminous urine, and the general loss of power bespoke some formidable organic disease. He was ordered a well-regulated nourishing diet. The emplast. ammoniaci c. hydrarg. was applied to the seat of the

FIG. 65

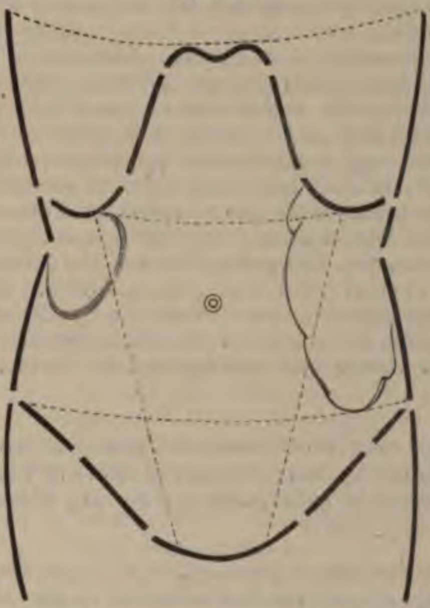


Diagram showing the situation of the tumors in the case of a patient with general cystic degeneration of both kidneys (after Bright).

tumor; and the uva ursi in infusion, and slight alkaline preparations, were directed to be taken. Under this treatment flattering reports were at first received, but the disease advanced, all the symptoms became worse, enlargement of the right kidney also became perceptible, the urine remained moderately coagulable (about a pint and a half in twenty-four hours), and he suffered a great deal of pain at the neck of the bladder, from the frequent passing of fibrinous coagula of a slight pinkish-yellow color, about an inch long, and apparently moulded by the urethra. His emaciation became extreme, and he had frequent returns of hæmaturia. From the middle of February he was completely confined to his bed, expecting death daily. In the first week of April he experienced some slight convulsive seizures, and fell into a state of coma for a few hours before his death, which occurred about the 10th of April.

Both kidneys presented most extreme specimens of vesiculated disease; the left was the largest, and was probably eight or ten times the natural size, while the right was at least six times the size of the healthy kidney. The whole appeared made up of a congeries of vesicles, from the size of a pigeon's egg to a pea; and the substance of the kidney was almost obliterated; nothing but a thin layer of secreting structure remaining, and that greatly altered from the natural texture. The pelvis of the kidney and the mamillary processes alone retained a tolerably healthy appearance; the lining membrane of the pelvis had no undue vascularity, and was perfectly smooth; the mammillary processes, though somewhat flattened, showed, when divided, the healthy organization; the ureters were healthy, but the renal vessels, particularly the veins, were large. The other viscera were healthy, and the bladder contained half a pint of urine.

The enormous size which the kidneys sometimes attain in this disease, and the abrupt termination of life, are illustrated by the following remarkable case recorded by Dr. Hare ("Path. Soc. Trans.," 1850–1, p. 131):

A man, æt. 46, was seen in January, 1850, for an attack of pleurisy. Under treatment, he recovered and returned to his business, until the 5th of March, when Dr. Hare was again called in. The night previously he had passed a considerable quantity of very bloody urine, which had, apparently, given him great relief from a constant pain he had in the left loin. On examination of the abdomen, which had lately become larger than usual, a tumor was found, extending from the cartilages of the false ribs of the left side to about an inch below the level of the umbilicus, and forwards to within about an inch of the median line. On percussion it was dull, and there was no interval of resonance between the tumor and the cartilages of the ribs; and the dulness on percussion extended upwards beyond the lower margin of the latter; the anterior border was rounded, but presented no signs of fluctuation.

In April, a considerable alteration was observed to have taken place in the tumor; it still felt solid and without fluctuation; the lower border extended an inch and half below the level of the anterior superior spine of the ilium. Its anterior border was deeply notched on a level with the umbilicus, and percussion was resonant at this notch, as also for some distance obliquely across the tumor. It presented very much the physical signs of a double tumor, or of two tumors; on placing one hand over the lower part of the abdomen, and pressing with the other against the left loin, although the tumor could be very slightly moved, it appeared to move as one mass. Dr. Bright, who also saw the case, spoke confidently as to its being "all kidney with intestine passing over it, and thus giving the appearance of two tumors;" there was also now a slight interval of resonance between the cartilages of the false ribs and the tumor. The urine, which had contained blood two or three times, was now clear.

On the 9th of December, he fell suddenly from his chair, convulsed and insensible; this was followed by sleepiness, numbness in both hands, and frequent twitchings.

On the 12th, when seen by Dr. Hare, he had a vacant expression, wandered a little, but answered sharply when spoken to; there were slight twitchings of the upper extremities. Pulse 72; feet and legs rather œdematous. The tumor appeared much the same as in April, except that it extended rather beyond the median line, and that the notch, at its anterior border, was less marked. Urine pale, without sediment, specific gravity 1008, containing one-tenth albumen.

On the 13th, he had two fits, somewhat similar to those on the 9th, and he died on the 16th, probably from the presence of urea in the blood.

Autopsy.—The left side of the abdomen was occupied by an enormous tumor which proved to be the left kidney, the intestines being pushed over to the right side. The tumor also extended under the intestines half-way across the right half of the abdomen; its upper surface was adherent to the diaphragm, and it had so compressed the spleen, that the latter formed, as it were, a cap to the kidney; the pancreas was carried forwards, and was adherent transversely to the anterior surface of the tumor, near its upper part; the descending colon, somewhat contracted, was likewise adherent to its anterior surface, but perpendicularly, so as to divide it into nearly equal portions.

The kidney measured 15¼ inches in length, 9½ in breadth, and about 23 in circumference, and weighed exactly 16 lbs.; it still retained somewhat the kidney shape, but its surface was uneven from the projection of different cysts. It consisted of one enormous congeries of cysts, varying in size from a small pea, to a cavity holding more than a pint of fluid; the larger cysts were at the surface, the smaller ones being about the centre; many of the smaller cysts projected more or less into the cavity of the larger ones; they presented different tints, from a dark purple, to a light straw-color (the latter much more rare than the former), according to the color of the contained fluids; the darker fluid was generally the thickest, and at the bottom of those cysts there was more or less of a dirty-red grumous-looking matter, which was wanting in those containing the lighter-colored fluid. The thickness of their walls varied generally in proportion to their size, the larger having the thickest parietes. No trace of the proper structure of the kidney was discoverable. The fluid, under the microscope, showed an immense number of blood-disks (more abundant in the darker fluids), some oil globules, exudative corpuscles, portions of the tubules of the kidney, and a considerable number of plates of cholesterine.

The right kidney presented incipient disease of the same kind, and was enlarged to double its natural size.

There was a slight hypertrophy of the heart, and a hernia above the umbilicus; the remaining viscera were natural.

In the following case described by Dr. Gray ("Path. Soc. Trans.," vol. vi. p. 267), the disease appeared to be occasioned by external violence: death was not preceded by cerebral symptoms, but by vomiting and hiccough, possibly of uræmic origin.

A man, æt. 40, much emaciated and anæmic, who had been very intemperate when young, was admitted into St. George's Hospital, March 7, 1855.

He dated the commencement of his present illness seven years ago, when he received a severe injury of the back; for some time after this accident he passed blood in his urine. He then partially recovered, but during the three following winters he often passed a small quantity of blood, and suffered much from pain in his loins. Five weeks before his admission into the hospital, he fell on his right hip, and then the hæmaturia and pains in the loins returned in an aggravated form. When first admitted, he was in a very weak and exhausted condition; the pulse was exceedingly feeble, and the urine was loaded with blood; vomiting and hiccough supervened, and he died exhausted on the 10th of March.

Autopsy.—There was extensive cystic transformation of both kidneys; and to such an extent had the disease proceeded that the natural structure of the glands could in no part be detected. The right kidney weighed 3 lbs. 10 ozs.; the left, 3 lbs. They were each about ten inches in length, lobulated on their surfaces, and composed of numerous separate cysts, varying in size from a pea to a small apple. The contents of some of the cysts were transparent and colorless, of others faint yellow, of others chocolate. The color in these last appeared to depend on blood-disks and their débris. The pelvis of the kidney and the ureter were not dilated.

All the other organs were natural, with the exception of the lungs, which were œdematous.

The primary lesion in this class of cases is possibly the same as in the congenital cases, and consists in a progressive occlusion of the ducts of the pyramids, leading at a later period to saccular dilatations of the tubes of the cortex, and finally to the formation of myriads of separate cysts. The cause of the occlusion, in most cases, is probably an interstitial inflammation, leading to the formation of contractile fibrous tissue. Thorn (Inaug. Diss. Bonn., 1882) found in one case that the inflammation had begun in the ureter and pelvis of the kidney, and had thence proceeded to the pyramidal portion. It is probable that the epithelium of the tubes also undergoes some active change, but of what nature is uncertain.[1] Conceivably the calibre of the ducts may be obstructed by plugs of coagulated blood. In Dr. Gray's case, just related, the latter explanation would appear to be a not impróbable one.

General cystic degeneration has evident affinities with the granular atrophic forms of Bright's disease, and probably requires a similar treatment.

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[1] *See* Cornil and Brault, Sur la Pathologie du Rein, Paris, 1884, p. 203. Chotinsky (Inaug. Diss. Bonn., 1882) asserts that, in the fœtal form, proliferation of the epithelium plays a part in blocking the tubes.

CHAPTER X.

CANCER OF THE KIDNEY.

CANCEROUS growths of the kidney may be *primary* or *secondary*. Primary cancer is attended by its proper symptoms and physical signs: it runs a distinctive course, and constitutes the cause of death. Secondary cancer, on the other hand, occasions neither symptoms nor physical signs: it is either a part-manifestation of a general cancerous cachexia, or an incident in the progress of primary cancer of some other organ; and its existence is usually unsuspected until the autopsy. It is therefore necessary to consider the two conditions apart, the latter indeed very briefly, as it has little, if any, clinical interest.

A.—PRIMARY CANCER OF THE KIDNEY.

The following description is based on an analysis of 68 cases, of which 64 were collected from various sources, and 4 contributed by myself. In all of them the disease was followed to its fatal termination, and the diagnosis verified by dissection after death.

The cases naturally fall into two groups—*children* and *adults;* and it will be desirable occasionally to distinguish the one class from the other. The first group embraces 25 cases under the age of ten years—indeed all, except 3, under five years. The second group includes 43 adults between the ages of nineteen and seventy.

MORBID ANATOMY.—The species of cancer found in the kidney is almost invariably the encephaloid (fungus hæmatodes).[1] It varies greatly in consistence and vascularity. In one instance it is described as being as soft as the milt of a fish; more commonly it is about as hard as human brain. The mass is seldom of uniform consistence throughout, and it is frequently the site of extensive hemorrhages. Cavities containing as much as a pint or more of clotted or fluid blood, or of blood mixed with cancerous detritus, have sometimes been found within the tumor.[1]

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[1] Two cases are reported in the Path. Soc. Trans., xxi. pp. 239, 241, in one of which Mr. De Morgan found the cancerous matter arranged in a villous manner; and in the other, recorded by Dr. Murchison (a case of villous disease of the bladder), the mucous membrane lining the pelvis and calices of both kidneys was studded with long villous processes.

Scirrhus is very rare in the kidney. Wilson mentions such a condition, but his description is vague. Rayer in one instance found a mass resembling mammary scirrhus in the midst of an encephaloid kidney:[2] and Dr. Walshe, among the unpublished drawings of Carswell, discovered one of scirrhus of the kidney: "the entire organ was converted into a gray-colored substance, somewhat transparent, and of the hardness of fibrous tissue. It was intersected in various directions by pale-colored bands which were opaque and firmer than the intermediate gray substance. It yielded only a small quantity of serosity on pressure, and presented few or no bloodvessels." (Walshe, loc. cit., 380.)

Colloid cancer has been occasionally found forming a part of an encephaloid kidney.

Epithelioma has not, so far as I know, been found in the kidney in the primary form in more than two cases. In one case published by Robin the right kidney of a man aged fifty-one was replaced by a large mass of adventitious tissue, of which part was soft and part hard. Both portions were composed of cells of epithelial character, most of them closely approaching the appearance of pavement epithelium, and attaining in the softer portions enormous dimensions. Some of the largest measured $\frac{1}{10}$ of a millimetre in length. None were found disposed in nests as in an ordinary cutaneous epithelioma.[3] The second case was observed by Waldeyer, and quoted by Birch-Hirschfeld in his "Lehrbuch der Pathologie," p. 1041.

Encephaloid invades the kidney sometimes in the nodular, sometimes in the infiltrated, form. It always begins in the cortical substance, and afterwards involves the pyramids. The epithelium of the renal tubules is first affected, and then the connective tissue.[4] The tunica propria is commonly thickened into a strong fibrous membrane.

When the whole organ is uniformly infiltrated, its natural shape and position may be tolerably preserved, even when it is enlarged to many times its original volume. But when a nodule grows from one end of the gland, leaving the remainder exempt, an irregular tumor is formed, which may assume shapes, and grow into situations very embarrassing for the diagnosis. The exempted portions rarely preserve their healthy state; the secreting structure wastes and degenerates; or it suppurates—though this is rare.

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[1] The late Dr. Hilton Fagge described a peculiar variety of cancer of the kidney, in which fatty degeneration had occurred, and which he styled, after Cornil and Ranvier, Carcinoma lipomatosum. (*See* Path. Trans., vol. xxvii. p. 204.)

[2] A similar case is reported by Dr. E. R. Townsend, Jr., of Cork. (Dub. Quart. Journ., xlv. 219.)

[3] Ch. Robin. Mémoire sur l'Epithelioma du Rein. Paris, 1855. *See* also Lebert, Anat. Pathol., ii. 351. Mr. Hoyle, in a patient who died in the Manchester Royal Infirmary, found epitheliomatous nodules in the kidney secondary to similar growths in the lungs, and in each organ describes cell-nests. (Journ. of Anat and Phys., vol. xvii. p. 509.)

[4] Cattani was able in one case to trace the development of the cancer cells from the renal epithelium. Dr. Norman Moore observed a similar appearance, and Dr. Sharkey traced the cancer cells to proliferation of the cell-lining of Bowman's capsule. (Path. Trans., vol. xxxiii.)

It is a marked characteristic of primary renal cancer, that it forms a tumor generally of large, often of gigantic proportions, which may stretch from the loin to the umbilicus, and from beneath the ribs to the pubes, and weigh many pounds. In 31 out of our 68 cases, exact information is given as to the weight of the tumor. In 16 children its average weight was 8½ lbs.; the smallest was 1 lb. 9 oz. and the largest 31 lbs.! In 15 adults the average weight of the tumor was 9½ lbs.; in one case the growth was about the size and weight of the natural kidney; in another it weighed 15 oz.; in two others it weighed 1½ lb.; in several it varied from 3 to 11 lbs., and in two attained a weight of 27 lbs. The enormous masses found in young children are really remarkable. In one example, recorded by Mr. Spencer Wells, a growth weighing between 16 and 17 lbs. was taken from the body of a child only four years of age ("Path. Soc. Trans.," xiv. 179).

The surface of an encephaloid kidney is usually soft, irregularly lobulated, and of unequal consistence. Not unfrequently it yields a deceptive sense of fluctuation, especially in certain spots.

Renal encephaloid is liable to the same accidents (degeneration, softening, suppuration, hemorrhage) as soft cancer elsewhere. In an instance mentioned by Bright, the softened mass burst into the peritoneum; in another, by Rayer, a cancer of the right kidney ulcerated into the duodenum; in a third, by Abele,[1] the disease broke through the abdominal parietes a fortnight before death, and formed a fungous ulcer through which a portion of the colon protruded and ultimately mortified.

The tumor generally contracts extensive adhesions to the surrounding parts. The colon is invariably found in front of the growth—though sometimes flattened and empty. The other abdominal viscera are thrust aside as the tumor enlarges: the small intestines are pushed over into the opposite flank. When the growth affects the right kidney, the liver is displaced to the left, often twisted on its transverse axis so that its upper surface takes a vertical direction and applies itself to the costo-abdominal wall. This distortion has been especially observed where, as in Döderlein's case, the growth protrudes from the upper end of the kidney, and makes its way into the right hypochondrium. When the tumor is constituted by the left kidney the stomach is pushed to right, and the spleen carried high up into the vault of the diaphragm.[1] The thoracic viscera are displaced upwards more or less according to the bulk of the tumor, and in various directions according to the side affected. Among other effects on the adjacent parts, caries of the vertebræ was twice found: more or less compression of the inferior cava generally exists towards the later periods, occasioning œdema of the legs and sometimes (though rarely) ascites.

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[1] Schmidt's Jahrb., Bd. v. S. 379

The pelvis of the kidney was found generally more or less involved, and, in the majority of cases, the ureter was permanently occluded by extension of the cancerous growth into it, or by blood-clots, or by the pressure of the main tumor.

The renal veins in several instances contained encephaloid matter; and in some of the cases it could be traced as far as the vena cava.

In the overwhelming majority of cases only one kidney was affected. Out of 67 instances which supply information on this point, the disease was confined to one kidney 60 times. In seven cases both kidneys were involved; but in three only of these did the disease appear to be primary on both sides; in the other four one kidney was the seat of primary cancer, which formed a tumor, while its fellow only contained small secondary nodules. In the 60 unilateral cases, each kidney was affected an equal number of times.

The primary disease in the kidney was associated with secondary deposits elsewhere in 31 out of 51 cases which give details on this point: in the remaining 20 cases all other parts were exempt. The most frequent seats of secondary deposits were the lymphatic glands in the hilus of the kidney, and the vertebral and mesenteric glands. These glands formed in some of the cases a large tumor, which—as in a case to be presently related—transcended the dimensions of the renal tumor, and greatly embarrassed the diagnosis. The lungs and liver were also often affected; the other organs more rarely; but instances are on record in which almost every conceivable combination existed. The following table exhibits the distribution of the secondary deposits in the 31 cases already alluded to:

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[1] In Case 2, reported a few pages further on, the position of the spleen was exceptional; it was carried downwards toward the iliac fossa in front of the tumor.

Kidneys alone affected	20 cases.
Secondary deposits found elsewhere	31 "

Seat of secondary deposits:

Lumbar, mesenteric and vertebral glands	15 "
Lungs	14 "
Liver	14 "
Suprarenal capsules	4 "
Omentum	3 "
Heart	3 "
Vertebræ and rib	3 "
Costal surface of pleura	1 "
Bladder, uterus, penis, and testicle—each	1 "

The infrequent association of primary renal cancer with cancerous deposits in the lower urinary passages, which this table shows, is somewhat remarkable, and is scarcely what one would expect, considering the close anatomical and functional relations of these parts.[1]

Etiology.—Renal cancer prevails at two distinct epochs of life—in early childhood, and in adult age. During adolescence the liability to it sinks to a minimum. Children under five years of age appear especially liable to renal cancer: 22 out of 67 cases occurred at this early period; 3 others between seven and ten years: the remainder were distributed pretty equally between the ages of nineteen and seventy. The annexed table shows more exactly the relation of age to the frequency of renal cancer:

	0–1 yr.	1–2 yrs.	2–3 yrs.	3–5 yrs.	7–8 yrs.	10 yrs.
25 children	2	6	6	8	2	1

	19 yrs.	20–30 yrs.	30–40 yrs.	40–50 yrs.	50–60 yrs.	60–70 yrs.	Above 70 yrs.
36 adults[2]	1	7	5	4	9	9	1

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[1] I had an opportunity of examining a man (æt. 40) who died at the Royal Infirmary with extensive cancer of the stomach, combined with primary cancer of the right kidney. The latter organ was wholly converted into an encephaloid mass. I could not detect any traces of secreting structure in it: the mass was somewhat smaller than the natural kidney, and about the same shape. The left kidney was quite healthy, and greatly hypertrophied. The right ureter was pervious, but shrunk to about half its usual size. The left ureter was somewhat more capacious than natural. The right suprarenal capsule was wholly converted into a cancerous mass. There was very extensive cancerous disease (in nodules) of the liver, and of the vertebral glands.

[2] In seven adults the exact age is not given.

The male sex is considerably more liable to renal cancer than the female. Sixty-six cases, in which the sex was distinguished, supplied 47 males, and 19 females. The preponderance of the male sex is not so great in childhood as in adult age. Of 24 children, 15 were boys and 9 girls; of 42 adults, 32 were men and only 10 women. The great disproportion between the frequency of renal cancer in men and in women may possibly be explained by the marked preference shown by cancer for the generative organs in the female.

The exciting cause of renal, as of other cancers, is wrapped in obscurity. In a few instances, a blow or fall on the loins was the immediate precursor and supposed cause of the first symptom; but the disease had doubtless already been in existence before the accident, though concealed. In a case mentioned by Manzolini ("Schmidt's Jahrb.," B. 94, S. 74), a boy was kicked in the left side; this was followed by hæmaturia for fourteen days. Shortly after, a swelling appeared in the left loin, which eventually proved to be an encephaloid growth of the left kidney.

Symptoms and Physical Signs.—The distinctive symptoms of primary cancer of the kidney are: *tumor in the abdomen* and *hæmaturia*. In every case in which the disease was the determining cause of death, one or both of these symptoms were present.[1]

Abdominal tumor is by far the most constant sign of renal cancer, and usually the earliest one noticed. Out of 64 cases there were only 3 in which a distinct intumescence could not be felt in the site of the kidney or thereabouts; and in these three there was hæmaturia. In the remaining 61 cases a tumor was easily ascertained to exist in the abdomen; and in all but three it was of such size and prominence that it could not escape the most cursory examination. It is noteworthy that in *all* the children a large—nearly always an enormous—tumor existed.

The tumor presents itself first in the anterior lumbar region, between the margins of the ribs and the crista ilii; it then grows forward to the umbilicus, upwards into the hypochondrium, and downwards into the iliac and inguinal regions: in extreme cases it fills the entire belly. The tumor may, or may not, be covered with a ramification of enlarged superficial veins. The colon, and sometimes a portion of the small intestines, lies in front of it. This position of the colon furnishes an important diagnostic mark of all renal tumors. Percussion over the tumor is dull, except where the colon intervenes.[1]

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[1] Lebert states that he has known an instance in which the disease ran a latent course throughout: but he does not say whether in that instance the renal disease was really the cause of death. In a case of primary cancer of both kidneys reported by Dr. Fleming to the Dublin Pathological Society (Dub. Quart. Journ., xliv. 235), the patient stated that he never had had hæmaturia, and there was no tumor traceable in the abdomen.

To the hand the tumor feels smooth or irregularly lobulated, with rounded obtuse margins. The lobulations are often of unequal hardness, and a deceptive sense of fluctuation may be felt in places, or in the tumor generally. In Langstaff's case, a distinct and persistent pulsation was perceptible in the tumor; and a similar phenomenon was noted in Bristowe's case ("Med. Times and Gaz.," 1854, ii. 395). The fixity of the growth is usually a marked characteristic.[2]

The second symptom in importance is hæmaturia. Details on this point are supplied in 59 cases. Of these, 28 exhibited no trace of hæmaturia throughout their entire course. In 31 cases there was hæmaturia; but in 5 of these, there existed other possible causes for it than renal cancer (calculi, Bright's disease, external violence). These figures, even with this abatement, do not sufficiently express the danger of relying too strongly on hæmaturia as a sign of renal cancer. In 6 instances hæmaturia occurred only for a few weeks at the beginning of the complaint, and then altogether ceased—the urine thereafter continuing normal. In one case there was hæmaturia for a short period at first, and none during the remaining four years of life. In another case (Case 1, shortly to be detailed) hæmaturia was present for some months, and at once disappeared on the voiding of a small calculus—it did not recur during the subsequent five years of life. In other cases hæmaturia did not appear until toward the last few moments of life—perhaps years after the detection of a tumor in the loin. The absence of hæmaturia seems to depend generally on the occlusion of the ureter, either by the pressure of the tumor or the extension of the disease into it.

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[1] This position of the colon was however not discovered in all the cases—generally, no doubt, from defective examination; but in some cases the detection of the gut may prove impossible, from its being compressed between the tumor and abdominal wall, and emptied of flatus. In doubtful cases, it might be of service to inject air per rectum, in order to inflate the collapsed gut.

The following remarks, by Bright, deserve to be borne in mind in searching for tumors of the kidney: "In those diseases," he says, "in which it (the kidney) most rapidly increases, the enlargement shows itself much more towards the anterior part of the abdomen than towards the loins, not only because the firm structure of this part is more calculated to conceal a tumor, but also because, in the other direction, it meets with less immediate resistance; so that it often happens, while we are examining the lumbar region with the greatest care, and obtaining but a doubtful evidence of fulness and hardness by the eye and by the touch, and by a careful comparison of the two sides, we can scarcely place the hand upon the anterior or even the lateral part without becoming at once sensible of the existence of a distinct tumor; and then, probably, by pressing that tumor backward, the other hand clearly informs us of its connection with the loins." (Loc. cit., 199.)

[2] In the Lancet for March 18, 1865, is an account of a case of movable kidney affected with malignant disease. The tumor was mistaken for an ovarian growth, and operation for its removal commenced. The intestines were all behind the tumor.

When hæmaturia is present, it is a sign of very great value, and its character and features deserve attentive study. As a rule, it is irregularly intermittent and profuse. It recurs at intervals of a few days or weeks, usually without any appreciable cause. The tumor is not, of course, insensible to external violence: and in more than one instance a blow or fall on the loin has been the immediate precursor of the appearance of blood in the urine. In some cases the hemorrhage is excessive, and followed by rapid anæmia and exhaustion, though this is rare. Generally the loss of blood is moderate, sometimes insignificant, and requiring the microscope for its detection. The formation of clots in the bladder, and their impaction in the urethra, is sometimes a source of severe suffering, and occasions excessive irritability of the bladder.

Other changes in the composition of the urine are sometimes found, but they are not distinctive. Of course, albumen always exists in the urine when it contains blood; more rarely albuminuria occurs independently of hæmaturia, from genuine Bright's disease affecting either the exempted portions of the cancerous kidney, or the opposite organ. Not unfrequently, epithelial cells from the pelvis of the kidney and ureter are found in the urine, mixed with the blood.

The presence of cancer cells in the urine is a sign which usually figures prominently in the catalogue of symptoms of renal cancer, but its value is very doubtful. In all the later cases, especially where there was hæmaturia, the urine was carefully examined for cancer cells, but without success. Rosenstein mentions a case in which a cancerous villus was actually found projecting into the ureter, yet no cancer cells could be detected in the urine during life. It is by no means an easy matter to identify cancer cells in the urine, in consequence of their similarity to the transitional epithelium of the pelvis and ureter. It must be further remembered, that any cancer cells which could find their way into the urine, must have escaped from parts of the growth which were broken down and degenerated; and to identify characteristic forms, in the ichorous detritus even of an external cancer, is more than I have ever succeeded in accomplishing: how much greater the difficulty, when that detritus has been further disintegrated by the action of the urine! In two examples of renal cancer, with hæmaturia, which I have had an opportunity of observing, repeated and careful examination of the urine failed to discover the presence of cancer cells or of cells which might be mistaken for them.[1]

[1] Mr. Moore believes that he succeeded in identifying cancer cells in the urine drawn after death from the bladder of a man in whose kidneys cancerous nodules were found; but his description rather accords with the appearances of the epithelial cells which are always freely detached from the vesical mucous membrane after death. (Med.-Chir. Trans., xxxv. 466.)

The other symptoms which have been noted in cases of renal cancer are less distinctive and constant than tumor in the abdomen and hæmaturia. The most important is pain in the hypochondrium and loin. This is sometimes an early symptom, and may show considerable severity. The pain is commonly intermittent; it shoots down in the course of the ureter to the inside of the thighs. It does not appear to be ever associated with retraction of the testicle. Pain is, however, wholly absent for long periods in a large number of cases: the tumor itself may be perfectly painless on handling, and give no inconvenience except from its weight and size.

Gastric symptoms—nausea, vomiting, anorexia,—are common, and in several cases they were noted among the earliest symptoms. In other cases, again, none of these existed: the appetite was excellent; in five cases (all children) it was even voracious.

The general health varied exceedingly. In the majority of cases, rapid emaciation took place, going on at length to an extreme degree, with failing strength, and yellowish discoloration of the skin. In other cases, many months, or even years (in adults), passed over after the detection of the tumor, before the health seriously gave way.

The cancerous tint is not often mentioned in the list of symptoms, but this may have been from the brevity of many of the reports.

The bowels are generally disordered when the tumor attains a large size; diarrhœa, or obstinate constipation prevails; or the two conditions alternate.

Towards the later periods, anasarca of the legs often sets in, and it may even extend over the whole body. Signs of constitutional irritation also present themselves, and become persistent. Life is at length worn out by gradual exhaustion of the vital powers: sometimes death is more suddenly induced by rupture of the tumor.

When there is no hæmaturia the urine is commonly normal; the healthy kidney becomes hypertrophied, and performs double duties. In no instance did uræmic symptoms arise.

The *duration* of the disease from the first appearance of symptoms to the fatal termination varied exceedingly. The duration was much shorter, as might have been anticipated, in children than in adults. Among the former, 19 cases are available for comparison: the mean duration was nearly seven months; the minimum was ten weeks, and the maximum "over a year." In adults (21 cases available) the disease continued on an average two and a half years; the extremes ranged from five months to seven years; 8 died under the twelvemonth, 7 under three years, 2 survived four years, 3 six years, and 1 seven years.

These numbers, as well as those having reference to the age of the patients, disagree with the statements current in books; and some of the numerous errors in the diagnosis of renal cancer may be traced to mistaken impressions as to the prevailing age and survivorship of patients so affected. The supposition of Walshe, endorsed by Lebert, that cancer of the kidney runs a more rapid course than other internal cancers, is not only unsupported by these large numbers, but the contrary is clearly established, namely, that, as a rule, death is longer delayed in renal cancer than in primary cancer of any other internal organ.[1] The reason of this tolerance must be looked for in the duplication of the organ, the facility with which one kidney undergoes a compensating hypertrophy when its fellow is disabled, and takes upon it the work of the pair; also the free room for enlargement which is afforded in the lumbar region, and the comparatively innocuous effects of displacement on the abdominal organs.[2]

The following examples will serve to illustrate the chief features of the disease.

CASE 1. *Encephaloid cancer of the left kidney; secondary cancer of left pleura.*—Mr. E., æt. 70, was visited by me with Mr. Jonathan Wilson, in March, 1868. He was suffering from an enormous tumor in the left flank. It appears that five years ago he suffered from profuse and repeated hæmaturia. After some months he voided a small calculus, and then the symptoms disappeared. He continued in good health for five years, and then came under treatment again for shortness of breath and general debility. Being somewhat fat and ventricose, he had not become aware himself of any tumor in the side; but when the abdomen was examined, a very large solid growth was discovered, filling the entire of the left half of the belly. How long this had been growing there was no evidence to show. The tumor occupied the left hypochondriac and lumbar regions, reaching forwards to the umbilicus and downwards almost to the crest of the ilium. Its front boundary had a rounded outline; its surface was wholly dull on percussion, even as far back as the spine; no bowel could be perceived in front of it. It felt hard, rigid, and perfectly fixed in its position. There was a moderate amount of ascites, with pleuritic effusion on the left side as high as the third rib; the other organs were healthy.

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[1] The mean duration of cancer of the pylorus, according to the combined statistics of Lebert, Herrich and Popp, and Valleix, is under a year: out of 71 cases, 48 died within the year, and almost all the remainder (23 cases) within two years. The majority of hepatic cancers terminate probably under eight months—certainly under a year (*see* Köhler, pp 308, 376). Walshe estimates the mean duration of cancer of the lungs at 13.2 months (loc. cit., p 348); and he thinks cancer of the brain rarely lasts over a year. (Ibid., p. 496.)

[2] Three exceptional cases of cancer of the kidney, which apparently lasted respectively 12, 14, and 16 years, will be found in the Lancet, 1877, i. p. 194, and p. 567. It must be remembered in connection with such cases that a benign tumor may exist for some time and then undergo transformation into a malignant growth.

The general condition was greatly depressed, the countenance sallow, the appetite almost lost. He lay continuously on his left side, and complained of great and constant pain in the left loin. There was no œdema of the feet or hands. The urine was scanty, high-colored, and charged with lithates; but there was no albumen or blood, nor had there been any for the last five years.

The patient was heavy and unwieldy, and unable to turn himself in bed; bed-sores formed, and he gradually sank from exhaustion, after having been under observation for a month.

Autopsy.—When the abdomen was opened, the tumor was found to be the left kidney, wholly changed into an enormous encephaloid mass. It was twelve inches long, eight broad, and five thick; it had an ovoid shape, and weighed eleven pounds. Its surface was smooth, and covered with a tough investment of fibrous tissue. In front of the tumor ran the descending colon, which was not adherent, but perfectly empty, and contracted to the size of the finger. On cutting open the tumor, it was found to consist of firm, yellowish-white, encephaloid matter, scattered through which were several masses of soft, clotted blood. There was no vestige of renal tissue. The ureter was healthy, but occluded by the pressure of the growth. The renal vessels were also healthy. The spleen was pushed up into the vault of the diaphragm, and lay above the tumor. The left pleura contained a large amount of sanguinolent fluid; on its costal surface were several cancerous nodules, as large as filberts. The other organs were healthy. The right kidney weighed seven and a half ounces, and was quite free from cancer. No calculous concretion was found in either kidney.

CASE 2. *Encephaloid cancer of the left kidney; unusual position of spleen and pancreas; calculi in the right kidney.*—F. M., a toy-dealer, aged 44, residing at Northwich, was admitted into the Manchester Infirmary in March, 1868. His illness began two years and a half ago with slight and temporary hæmaturia. Fourteen months ago more violent hæmaturia took place, which has continued more or less ever since. His medical attendant discovered a lump in the left side a twelvemonth ago, and since then he has gradually become weaker and thinner, and the lump has steadily increased in size.

On admission he was extremely emaciated, countenance of a greenish sallow appearance, drawn and suggestive of suffering; tongue red and dryish; p. 94, r. 24. No œdema of any part. The abdomen was much enlarged, especially on the left side, and on palpation a large solid tumor, as large as a man's head, was felt on the left side of the abdomen, occupying the epigastric, hypochondriac, and lumbar regions. The limits of the tumor, as ascertained by palpation and percussion, were as follows (*see* Fig. 66). The anterior margin could be traced from a little to the right of the ensiform cartilage, running downwards an inch and a half to the right of the middle line. About an inch above the umbilicus, the outline twined abruptly to the left, crossed the middle line, and then descended obliquely into the iliac fossa, as low as the crest of the ilium. Upwards, the growth extended beneath the ribs, bulging these out, almost as high as the nipple. Posteriorly, the growth occupied the whole lumbar region, and caused a marked fulness in the site of the kidney. The area of the tumor was dull on percussion, except in the epigastrium, and along the left costal margin, where a tympanitic note indicated that the stomach lay in front of it. The descending colon could easily be traced in front of the tumor—it was often loaded with masses of scybala. Over the lower part of the tumor, in the iliac fossa, there lay a detached portion, resembling, both in shape and in feeling, a somewhat enlarged spleen; this portion was freely movable upon the main tumor, and had no intestine in front of it. The upper part of the tumor in the epigastrium had a nodular tuberous feel, and was covered by bowel. Along the costal margin a large artery could be felt pulsating on the surface of the tumor; this artery communicated a distinct thrill to the finger, and was the seat of a loudish systolic murmur. The general surface of the tumor was smooth and tensely elastic —not fluctuating, but different parts varied sensibly in their degree of softness and hardness. The growth was immovably fixed in its position, and meandering veins coursed over the skin, covering its outer and more prominent portions. There was also an abundant varicocele on the left side, and the veins of the penis were very large and tortuous.

FIG. 66.

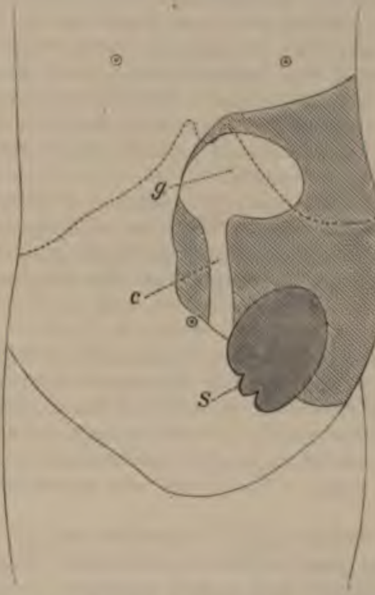


Diagram showing the position and relation of the tumor in the case of F. M.: *g*, stomach; *c*, colon; *s*, spleen.

The urine contained blood, partly in small, round, or filamentous clots, and partly mingled with the urine. There were no casts, and the quantity of albumen was not greater than the blood accounted for.

The organs in the chest were healthy, but they were considerably displaced upwards by the pressure of the abdominal tumor. The heart's apex beat in the fourth interspace, and the cardiac dulness mounted as high as the second rib. The liver was also pushed upwards; its upper margin corresponding with the fifth rib in the vertical line of the nipple. The patient complained of a good deal of pain of a gnawing character in the left loin and in the groin.

The patient remained in the Infirmary for six weeks. During this period the physical signs underwent no marked change; the tumor slowly enlarged, and the strength and flesh continued to decline. The urine always contained more or less blood, but the quantity was never really large, and very often it required the microscope to detect it. It was noticed that there was generally more blood in the urine after prolonged manipulation of the tumor. The bowels were exceedingly torpid, and required the frequent use of enemata for their relief. Beyond the use of these and of anodynes to procure sleep and allay the pain, no remedial means were attempted.

After leaving the Infirmary, he returned to Northwich, and placed himself under the care of Mr. Williams, of that town. He continued to sink very gradually as the tumor enlarged, and died on October 8th —five months after his discharge from the hospital, eighteen months after the first discovery of the tumor, and three years after the first appearance of hæmaturia.

Careful inquiries respecting the hæmaturia elicited the following particulars: Blood first appeared in the urine three years before death. For three weeks at that time pure blood was repeatedly voided; then for a period of twelve months no more blood was seen. Again about a pint of blood was voided, and after this it continued in greater or less quantity until his death. The blood was nearly always more or less clotted.

I went over to Northwich to make the *autopsy* with Mr. Williams. The emaciation had reached the most extreme degree compatible with life; the muscular tissue had almost vanished. Our astonishment was great to find the abdomen sunk so as scarcely to constitute a notable tumor. This had arisen apparently from the oozing out of the tumor of a considerable quantity of a bloody fluid, which we found in the peritoneal cavity. When the belly was opened, we found a large subglobular mass, occupying the whole of the left side. This proved to be the left kidney, converted into a mass of soft cancer. Overlapping its upper end was the empty stomach, and along the great curvature of this viscus coursed, with very tortuous windings, an artery as large as the radial (right gastro-epiploic). This was evidently the source of the pulsation felt during life at the costal margin. Riding freely on the lower and inner (or right) border of the tumor and the adjacent portions, lay the spleen, forming a flattened oval cake, 7 inches long by 4 inches wide. This was the movable spleen-like body felt during life near the iliac fossa. Between it and the stomach stretched the transparent layers of the gastro-splenic omentum. The colon, contracted and empty, passed in front of the inner portion of the tumor, under the spleen, and again in front of the lower part of the tumor, crossing it obliquely near its right margin. The body and tail of the pancreas ran horizontally right across the tumor, midway between the border of the stomach and the spleen, behind the folds of the gastro-splenic omentum.

The tumor constituted a somewhat flattened and elongated sphere, in parts with a lobular or tuberculated surface, and in part smooth. It occupied the entire vault of the diaphragm on the left side, the epigastrium, and the left lumbar region as low as the crest of the ilium. It was immovably fixed here, and adherent to the diaphragm and to the soft parts of the loin. The spleen and stomach were not adherent; the pancreas and omentum were loosely adherent. The tumor, in the epigastrium, extended two inches beyond the middle line, pushing aside the liver and pressing on the vena cava and its branches. Some of the mesenteric veins were also compressed and enormously distended; the varicocele was quite effaced after death. The tumor weighed six pounds. At its lower part the vestiges of the kidney could be recognized—still preserving the outline of the gland. The ureter, somewhat smaller and more transparent than natural, but still pervious, could be traced into the remains of the pelvis. On dividing the tumor, the mass was found to consist of a soft cellular encephaloid matter, deeply infiltrated in parts with blood. The scanty remnants of the kidney, in the form of a thin layer of indurated cortical substance, was stretched over the lower part of the tumor. The pelvis consisted of three or four communicating loculi, filled with a yellowish, gelatinous material.

The right kidney was of its usual dimensions, and its substance healthy; but the infundibula were dilated, and contained eight phosphatic calculi, varying from the size of a horse-bean to that of a hempseed, together with a multitude of smaller granules of the same nature. All the other organs of the body were healthy, and there were no secondary cancerous deposits anywhere.

The positions of the spleen and pancreas were quite exceptional in this case. The spleen is usually carried up above the tumor into the vault of the diaphragm: here it lay in front, and was pushed downwards into the iliac fossa. The pancreas is generally left undisturbed in its normal situation; but in this case it was stretched in front of the tumor. Both these conditions were probably due to the morbid growth having commenced at the upper end of the kidney.

It is almost certain that the hæmaturia in this case was not altogether derived from the left (or cancerous) kidney. The earlier and more profuse bleedings were in all probability so derived; but the scantier hemorrhage of the later periods could not have had this source, because the pelvis of the left kidney when examined after death only contained a clear yellow fluid, and the secreting substance of the organ was completely destroyed. The scantier but more constant hæmaturia, from which the patient suffered while in the Infirmary, was, doubtless, derived from the right kidney, in the infundibula of which a number of calculous concretions were found after death. In further evidence of this it should be mentioned, that the urine, while the man was under observation, always contained small calcareous particles composed of phosphate of lime—exactly similar in composition to those found in the kidneys at the autopsy.

A typical example of infantile renal cancer was shown to me by Dr. Lloyd Roberts. The following are his notes of the case:

CASE 3.—W. A. McE., aged six months, was sent to me by Dr. Cran, of Salford, on May 1, 1871, suffering from abdominal tumor. The mother stated that at the child's birth the nurse thought he had a "full stomach." When a fortnight old he suffered from a severe attack of abdominal pain and flatulency, requiring the attendance of Dr. Cran. After this he remained pretty well until the age of three months, when it was observed that his abdomen was larger than it ought to be; and it continued from this time to enlarge. The child was subject to frequent attacks of diarrhœa during the earlier months of his life, the stools appearing like "boiled moist cabbage;" but latterly four or five days would elapse without any evacuation, the motions being dry, hard, and yellow. Up till the time of death he passed water freely; it was always clear and free from blood. The appetite was voracious, but he never seemed to rest until he had vomited his food. He suffered much from thirst.

On the first of May the following was his condition: He was much emaciated. The veins of the abdominal wall were much distended. The abdomen measured 21 inches over the umbilicus, and was almost entirely filled by an immovable tumor of somewhat irregular shape. There was universal dulness over the abdomen, with the exception of the left hypochondriac and hypogastric regions, where a clear bowel-sound was elicited on percussion. On the 7th of July, the abdomen measured 24 inches, having increased three inches in a little over two months. The child died on 14th July.

At the *autopsy*, the liver was found thin and stretched over the surface of a large tumor, which was found occupying the entire abdomen, except the left inguinal region, into which the intestines had been pushed. There was no fluid in the peritoneal cavity. The liver, which was pale, but seemed otherwise healthy, was connected with the tumor by loose cellular adhesions, which were easily separable by the fingers, so that the entire tumor was removed without the aid of a knife. It was found to be a tumor of the right kidney, and weighed 5½ lbs. The growth was irregularly kidney-shaped, and traces of renal structure were detected at its posterior part. On section, it was found chiefly composed of soft, brain-like structure, with several large cysts containing fluid, which on microscopic examination was seen to be crowded with caudate and polygonal cells. The same bodies, in a fibrous-looking stroma, were detected in the solid portions of the tumor. The left kidney was rather larger than usual, but was healthy in structure. No cancerous deposit was found in any of the other viscera.

CASE 4. *Primary cancer of the right kidney, and of the lymphatic glands in the hilus. Secondary cancer of the liver, left lung, and suprarenal capsule* (from the notes of Dr. Renaud).—Hannah Hilton, æt. 59, a married woman, who had borne children, the last from eight to nine years ago, ceased to menstruate six years ago.

She first noticed a small and hard tumor in the right iliac space two years since, which made very little progress, and gave no pain or inconvenience for many months. She came under treatment in the early part of December, 1845, for a chronic diarrhœa, which had for some time past baffled all remedies. The evacuations were most copious, of a dirty olive color, passed without pain or accompanied with tormina. This ultimately yielded to the sulphate of copper.

I first examined the tumor about twelve months ago. It was painless, hard, and not bigger than a fœtal head, rising a little out of the right pelvic region. About a month from this, the tumor began to be painful and to increase. A feeling of crumpling parchment was noticed at its inner and lower portion. Shortly afterwards it began to extend upwards and backwards, in the direction of the loin; there were flying pains also. The uterus, examined manually, was found free from the tumor, and apparently healthy. The color of the skin was somewhat dirty, and this, together with the crumpling, were thought sufficiently suspicious to warrant a belief that the real nature of the disease was malignant degeneration of the right ovary.

I now lost sight of the case until three weeks prior to death, when I discovered that the tumor had gradually extended itself backwards, and that four weeks ago it began rapidly to grow, and spread in all directions, causing great pain and watchfulness. The woman emaciated very fast, was of a deep and dirty-brown color, had sunken eyes, and a look of suffering. The nature of the disease remained no longer doubtful, for the nodular portions of the fungus hæmatodes could be most distinctly felt beneath the abdominal walls. The feeling of crumpling was also more general, as also an occasional gurgling as of air, in the intestines. There had been no uterine hemorrhage, no difficulty or pain in passing urine, and nothing unusual in the character of the secretion. Opiates relieved the pain greatly. She died on February 17, 1847.

Autopsy.—Body greatly emaciated. In the abdomen there was a large fungoid tumor, extending quite across and to the right side and loins; passing obliquely over it was the colon, which was partially adherent, as were also some of the small intestinal folds. The tumor was not at all adherent to the interior of the abdominal walls. The uterus and ovaries were quite free from any disease, and were merely bound together with false membranous bands. The tumor had no pedicle, and though most carefully removed there were no connections found other than such as had been set up through peritoneal irritation. The abdominal cavity did not contain any dropsical effusion. The entire mass being removed, together with the liver, to which it was adherent, the right kidney was found so entirely degenerated into encephaloid matter, and so closely incorporated with the tumor, that nothing but a most careful dissection could have detected its true nature. It was enlarged to double its usual size, and no vestige of its proper structure remained; the vessels were, however, found entering the hilus; and the suprarenal capsule, also affected with encephaloid cancer, was in its usual position, and, in size and shape, bore a resemblance to a very large chestnut.

In the large tumor, which appeared to have its origin in the lymphatic glands of the hilus, were some cysts, filled with a grumous matter or with a semi-transparent jelly-like substance. The great mass was a homogeneous and soft cancer, breaking down in most parts, but in some places as hard as cheese. The tumor was rounded, and about four inches thick, where it lay in the loin on the right side, and gradually became more thin toward the left margin, where it dipped beneath the stomach, surrounding the aorta and vena cava, in one portion of which cancerous matter was found. The extreme breadth of the tumor was nine inches.

The lower margin of the liver was cancerous where the tumor came in contact with it, and some other small cancerous tubera were found on its surface. The gall-bladder contained many calculi. On the surface of the lower lobe of the left lung were several tubera, and one as large as a small apple. The heart, left kidney, spleen, right lung, and other parts were healthy, and free from all traces of disease.

Dr. Hounsell has kindly furnished me with the notes of the following hitherto unpublished case. It illustrates the usual features of the disease as it appears in children.

CASE 5. *Encephaloid disease of the right kidney in a child* (from the notes of Dr. Hounsell, of Torquay).—Richard Bradford, four years of age, was admitted as out-patient of the Torbay Infirmary about ten days before his death. He was suffering from a large tumor occupying the umbilical, right hypochondriac, and lumbar regions. Its surface was dull on percussion, and the dulness was continuous with that of the liver. The child was of a sallow appearance, and much emaciated. The tumor had been detected three months previously, and had grown rapidly. Hæmaturia had been noticed shortly before the discovery of the tumor.

Autopsy.—The tumor was found to involve the right kidney; it weighed 10 lbs. 14½ ozs.; it was smooth on the surface, and to the touch felt firm in some places, and soft, almost fluctuating, in others. Near it lay a supplementary tumor about the size of an orange. On cutting open the tumor it was found to consist of soft brain-like substance, containing two or three large cysts filled with about half a pint of dark fluid. The tumor sprang from the upper portions of the kidney, and had absorbed all the organ except two of the pyramids, which remained intact. The ureter, the left kidney, and all the other organs were healthy. The liver was adherent to the tumor, and the ascending colon ran along its lower border, and could not be detected in front of the growth during life.

CASE 6. *Enormous malignant disease of the left kidney* ("Lancet," 1856, i. 626).—J. B., aged six years, was admitted into the Middlesex Hospital, under Dr. Hawkins, May 29, 1855. J. B., born of healthy parents, was one of a family of ten children, of which five were still living, the others having died of acute infantile diseases. When the child was six weeks old his mother noticed that both left extremities were larger than the right; the skin was looser, and the muscles she describes as being less firm than those of the opposite side. She was so struck with the difference that she consulted a medical man about it.

At three years of age the child had whooping-cough, and shortly after measles, but he never had scarlet fever. The abdomen, the mother believes, was always rather larger than could be considered natural, but this had not been to a marked extent. With these exceptions, the child had fair health until the middle of April, 1855 (six weeks before admission), when he was suddenly seized with sickness; and from his appearance the mother believed him to be very ill, and though far better on the following day, so much as to be able to walk out of the house, he did not regain his appetite for about a week. During this illness the mother accidentally discovered a tumor in the upper part of the left side of the abdomen. It then appeared to be almost circular, and about two inches in diameter. It was not perceptible to the eye, but its lower margin could be distinctly felt; it was very hard, but not painful, nor did moderate pressure cause any inconvenience. She believes that it gradually increased in size after she first discovered it, till she brought the child to the hospital, and during this time she noticed that he had quite regained his appetite, which had, in fact, become voracious, and though he appeared fatigued after moderate exercise, he was able to walk without effort.

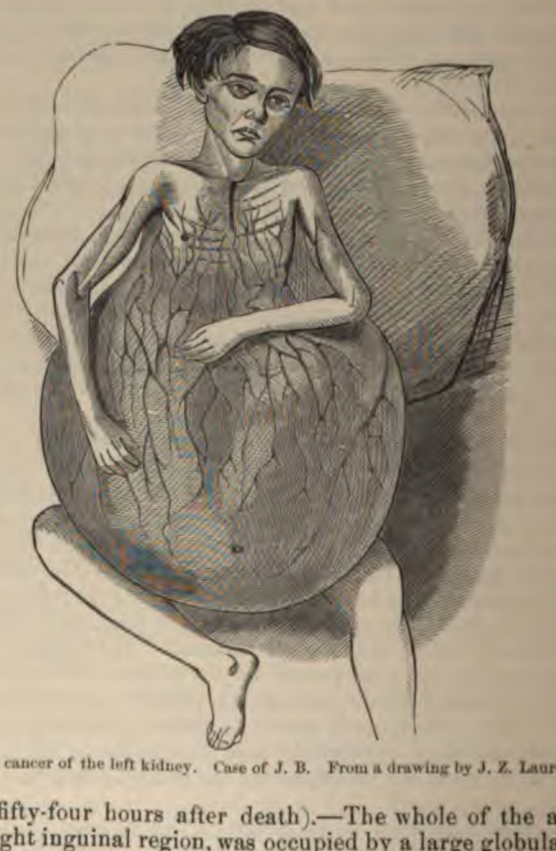
State on Admission.—Rather emaciated; abdomen very much swollen, especially on the left side, where the veins were enlarged and tortuous; the left extremities were considerably larger than the right, owing to the soft parts being much firmer and the muscles apparently better developed; there was, however, no difference in length. Upon manipulation, a tumor could be felt, of somewhat globular form, about three inches in diameter, occupying part of the left hypochondriac, left lumbar, and umbilical regions; its lower margin was well defined, but its upper boundary could not be ascertained—the dulness on percussion, which was complete over all parts of the tumor, being there continuous with that of the spleen. The patient ate, drank, and slept well, was able to sit up the greater part of the day, walked frequently up and down stairs, and did not complain of pain.

From this time the patient continued under observation until his death, a period of nearly twelve months. The tumor continued rapidly to grow, until it attained enormous proportions. On the 1st of August, the following note was taken: "The tumor extends half an inch below the umbilicus, and about the same distance to the right of the mesian line; the abdomen generally is much more swollen, and the veins are much larger. The patient walks about the garden for an hour or two every day, and though taking a large quantity of food, and eating very frequently, is daily becoming more emaciated. He appears to suffer no inconvenience, except that caused by the bulk of the tumor, the large size of the abdomen being such as to impede progression. He complains of thirst, and evinces a desire to drink frequently of cold water. The bowels act with regularity; and the urine, which is frequently voided, and in quantity rather above the natural standard, presents no abnormal appearances."

The patient continued to go about till September, and to walk up and down one flight of stairs to and from the ward without assistance. About the middle of the month, after having spent some time in the garden of the hospital, he fancied himself unable to get back, and was then for the first time carried upstairs. After this, he was almost constantly confined to his bed. The tumor gradually increased in size until his death; for some time previous to which, indistinct fluctuation could be felt in some parts of it. The abdomen, about the middle of December, measured in circumference 36 inches, and at the end of March upwards of 42 inches. For the last two months he suffered much from dyspnœa; and for the last three weeks, had constant orthopnœa, and daily increasing œdema of the left leg. The appetite, however, remained inordinate till the last; and the bowels, which had continued to act regularly till within a short time of his death, had recently become somewhat constipated. He sank gradually, and died April 7, 1856.

The annexed drawing of the patient was taken shortly before death, by Mr. J. Z. Laurence, and kindly placed by him at my disposal.

Fig. 67.



Enormous cancer of the left kidney. Case of J. B. From a drawing by J. Z. Laurence.

Autopsy (fifty-four hours after death).—The whole of the abdomen, except the right inguinal region, was occupied by a large globular tumor, anteriorly firmly adherent to the parietes, and covered by peritoneum; posteriorly, lying in contact with the psoas muscle; the small intestines were thrust down to the right inguinal region; the spleen and liver were driven upwards into the thorax; the whole of the transverse colon was firmly adherent to the tumor; and a portion of the descending colon, which ran along the front, was for a short distance embedded in it. The tumor, when removed from the body, weighed thirty-one pounds. Traces of kidney structure could be recognized, as if spread out over the entire substance; large masses of medullary cancer were visible on its surface. Upon section, the centre was found to be occupied by several pints of dark, thick fluid, floating in which were several fragments of the broken-down cancerous mass; the more solid portions varied in consistence from that of firm medullary cancer to gelatinous matter in a semi-fluid state, large masses of it being found in every stage of degeneration; the kidney on the opposite side was much enlarged. No cancerous deposit was found in any of the other viscera.[1]

DIAGNOSIS.—We have seen that in nearly all cases of primary cancer of the kidney, a palpable tumor exists in the flank. If profuse hæmaturia coexist with such a tumor, scarcely a doubt can remain as to the seat and nature of the disease.[2] But when there is no hæmaturia, the diagnosis becomes more difficult; indeed, there is scarcely any morbid condition which has been so frequently misapprehended. Renal cancer has been generally mistaken for enlargements of the surrounding organs—of the liver, spleen, ovary, or uterus; but sometimes for ascites, aneurism of the aorta, or perinephritic abscess. It has also been mistaken for tumors of the kidney of a different character—for pyonephrosis, hydatid, cystic degeneration, and hydronephrosis. Some of these errors were doubtless unavoidable; but most of them arose from an imperfect knowledge of the diagnostic marks of renal tumors, and from the undue weight attached to the absence of hæmaturia. As a positive sign, associated with abdominal tumor, hæmaturia—profuse, spontaneous, and recurrent—is of the highest significance; but its absence signifies comparatively little. In nearly half the cases collected by me, hæmaturia was wholly absent from the first to last; and in those cases in which hæmaturia was noted, intervals of many weeks or months elapsed in several of them, during which the urine was perfectly normal.

In those numerous cases, therefore, in which the observer derives no help from the examination of the urine, he must rely on his skill to ascertain the anatomical relations and nature of the abdominal tumor. In prosecuting this inquiry, he will especially endeavor to eliminate tumors of the liver, spleen, and ovaries—these being, from their comparative frequency, the most likely to lead astray.

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[1] Some further particulars of the post-mortem appearances in this case are supplied by Dr. Van der Byl (Path. Soc. Trans., vol. viii.).

[2] The coexistence of these two symptoms is not, however, absolutely diagnostic of renal cancer. In a case of enormous enlargement of the spleen (leucocythæmic) in the Manchester Infirmary, there was profuse hæmaturia for several days. After death, some months subsequently, the kidneys and bladder were found perfectly healthy.

If the intumescence occupy the right side, it may be distinguished from hepatic tumor, especially when not very large, by the possibility of tracing its upper limits below the margins of the ribs; the side of the hand can generally be so inserted at the edge of the ribs, that the tumor can be clearly felt to lie below it, and the liver above it. Along this line a coil of intestine usually lies, and yields a tympanitic sound on percussion. This sign is lost, however, when the renal growth contracts adhesions to the under surface of the liver; also when it projects disproportionately into the right hypochondrium, and displaces the right lobe of the liver. When this is the case, assistance may be obtained by feeling for the thin margin of the liver as it lies applied to the abdominal wall. Another important sign in such a case is the position of the colon. Hepatic tumors have no intestine in front of them (unless there be malposition of the viscera), and yield a dull note over their entire surface. Renal tumors, on the other hand, have the ascending colon in front, passing obliquely from below upwards and to the left; and the passage of flatus along the gut, or the clear percussion note over it, will rarely fail to indicate its position.

A splenic enlargement is distinguished by the following signs: absence of the descending colon in front; its rigid, somewhat thin borders (not rounded); its extension upwards under the ribs; its mobility; generally, a tympanitic note is obtained in the extreme left lumbar region; often, on deep percussion, a bowel sound is perceived through its substance, which is not thick (a renal tumor is absolutely dull on the deepest percussion); antecedent history of ague or remittent fever, or evidence of leucocythæmia on examination of the blood; the direction of the enlargement is downwards and inwards to the epigastrium and umbilicus, and not toward the iliac fossa. It also rises higher toward the axilla than a renal growth. When the latter rises from the upper and forepart of the kidney, and pushes forwards and upwards rather than downwards, the diagnosis becomes very difficult, and depends mainly on the absence or presence of the colon in front of the enlargement, and hints derived from the previous history or the state of blood on microscopic examination.

When the tumor presses forwards and downwards toward the umbilicus and the pubic and iliaç regions, it is apt to be mistaken for ovarian tumor. The commemorative symptoms may here yield valuable information, though the statements of patients on such points are always to be accepted with reserve. An ovarian growth begins in the iliac fossa, and ascends; a renal growth begins in the flank between the ribs and the crest of the ilium, and descends. An ovarian tumor has no bowel in front of it, and the bowels are pushed into the lumbar region, where a clear sound can be elicited—exactly in the spot where the dulness is most complete when the tumor arises from the kidney.[1] This last sign also serves to distinguish uterine from renal enlargements.

An encephaloid kidney can only be confounded with ascites when it is extremely soft, and fills the entire abdomen. The two conditions may be distinguished by the circumstance, that in ascites *both* flanks are dull, whereas in renal tumor one is dull and the other resonant.

When the tumor has been satisfactorily made out to be connected with the kidney, there still remain difficulties in deciding its nature. Malignant growths generally give a distinct impression of their solid structure. This distinguishes them from hydatid, purulent, and hydronephrotic cysts; but the consistence of the tumor is often very difficult to appreciate: if it be small and deep-seated, and the abdominal walls thick, the sense of fluctuation in a fluid cyst may be exceedingly obscure; on the other hand, encephaloid tumors sometimes yield a quasi-fluctuation which is very deceptive. In these doubtful cases, the presence of pus, or blood, or hydatids in the urine, of rigors, of nephritic colic, or of cancerous cachexia, supplies hints which incline the judgment in this or in that direction.

PROGNOSIS.—The ultimate termination is, of course, always fatal. In judging of the probable survivorship of the subjects of renal cancer, the age of the patient is of great importance: the mean duration of the disease is at least three times as great in adults as in children. There is, however, nothing like exact proportion observed in this respect. In a girl of twenty-one, whose case is described by Langstaff, the disease lasted (with hæmaturia) for six years. Contrary to what might have been expected, the occurrence of hæmaturia does not appear to hasten the final catastrophe: the mean duration is almost exactly the same in the hemorrhagic cases, as in those in which the urine was throughout normal.

The disease appears in some cases to become dormant for a while, making no appreciable progress for many months. In an instance of this kind recorded by Dr. Brinton, the stationary condition (which Dr. B. had flattered himself might pass into permanent obsolescence) came suddenly to an end, with death of the patient, through copious hemorrhage into the tumor. ("Brit. Med. Journ.," June 13, 1857.)

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[1] A case is reported by Dr. Greenhalgh ("Tumors Complicating Pregnancy," St. Barth. Hosp. Reps., vol. i. 85), in which a tumor supposed to be ovarian acted as a complication in two pregnancies; and the propriety of removal was about to be entertained when the patient again became pregnant. She died without obvious cause three weeks after delivery at the full term, and the autopsy showed that the supposed ovarian tumor was really the left kidney in a very advanced stage of cerebriform disease. It weighed 27 lb. 3 oz.

TREATMENT.—The management of a disease so hopeless is a melancholy duty. When the tumor is painless, and the urine natural, there is little for the practitioner to do beyond placing the patient in favorable hygienic circumstances. When the tumor is tender, or there are signs of local inflammation in its vicinity, warm baths or emollent applications may be used from time to time. It may be doubted whether it is prudent to interfere with a moderate hæmaturia. The losses of blood do not on the whole act disadvantageously. When, however, the hemorrhage becomes excessive, means must be used to control it. Ice may be applied to the tumor, and acetate of lead or gallic acid administered internally. The clots which form in the ureter and bladder sometimes occasion the most poignant suffering by blocking up the urethra, and causing retention of urine. The impacted masses should be pushed back into the bladder by means of the catheter, and the coagula broken up by washing out the organ with warm water.

As the disease advances, severe constitutional irritation sets in, which requires to be palliated by opiate and other anodyne medicines.[1]

B.—SECONDARY CANCER OF THE KIDNEY.

Secondary cancerous deposits occur in the kidneys, in the form of nodules varying from the size of a pea to that of a marble or walnut. Ten to twenty such nodules are not unfrequently found scattered through the cortical substance: the intervening renal tissue shows no sign of disease; the urine is normal, and no pain or other symptom betrays their presence during life, The following case offers an example, marked by some very unusual incidents, of extensive cancerous disease of the urinary organs, involving primarily the bladder and its vicinity, extending thence to both kidneys, of which the right was undergoing sacculation from compression of the corresponding ureter by the cancerous mass at the base of the bladder.

In January, 1862, I was requested by Dr. Crompton to see with him a shopkeeper, aged 38, who was then suffering from hæmaturia and paralysis of the bladder. The patient gave the following account of himself: Three years previously, without known cause, he had an attack of hæmaturia, accompanied with excessively frequent micturition, pains in the back and bottom of the belly, but without vomiting or retraction of the testicle. These symptoms passed off, under medical treatment, in two months, and (apparently) complete recovery soon ensued.

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[1] A curious case is reported in the Philadelphia Medical and Surgical Reporter for 1861, p. 126. A man of 57 had had a tumor in the right hypochondrium for six years. It was supposed to be "cystic disease" of the liver; and his surgeons deliberately proceeded to remove it by operation. The tumor (which weighed 2½ lbs.) was accordingly removed, but on examination it proved to be the right kidney, wholly converted into an encephaloid mass. The patient survived fifteen days.

After an interval of three years, during which the patient's health continued in every respect undisturbed, the present attack abruptly commenced. The patient was seized, six weeks before my visit, with violent pains in the loins and hypogastrium, accompanied by painful and excessively frequent micturition and bloody urine. All these symptoms came on simultaneously. There was neither sickness nor vomiting. The attempts to void urine were incessant—every ten or fifteen minutes during the day, and so constant at night that the patient scarcely obtained any sleep. Matters continued thus for three weeks; the patient, meanwhile, did not keep his bed, and he attended, as well as he was able, to his duties in the shop.

But a new train of symptoms now showed themselves. The incessant micturition was succeeded by a total inability to empty the bladder, and the legs and belly began to swell rapidly. At this conjecture Dr. Crompton's aid was obtained. On examining the patient he found considerable ascites, anasarca of the lower extremities, and retention of urine. Three pints of a sanguinolent urine were immediately withdrawn by catheter from the distended bladder; the patient was directed to keep his bed, and treated with alkaline diluents and nightly sedatives. Great relief followed this treatment, but the patient still continued unable to void a drop of urine spontaneously, and catheterism had to be practised twice a day.

His condition at the date of my visit was as follows: There was extreme pallor of the surface; considerable emaciation; no pyrexia; the tongue was moist, slightly furred. The legs were no longer œdematous, but considerable ascites still remained. The bladder was distended almost to the umbilicus; there was no pain, and the loins were not sensitive to pressure; nor was there any tumor to be felt in the renal region; the movements of the patient were active, and he was cheerful and lively.

About a quart of bloody urine was removed by catheter. A little pure blood came through the instrument first, then almost clear urine, and as the bladder became empty, the urine again became ruddy, the last few drops being almost pure blood. Dr. Crompton stated that a little bleeding always followed the morning and evening catheterism.

A careful examination of the urine yielded the following: It was feebly alkaline from fixed alkali (derived from medicine); specific gravity 1007; on standing, the blood-corpuscles subsided, and formed a very red, slightly clotted layer, at the bottom of the urine-glass. Under the microscope there were found, in addition to the blood-disks, a few corpuscles with cleft nuclei—probably pale blood-corpuscles—but no renal elements—neither epithelium, nor casts, nor any suspicious (quasi-cancerous) cells of any sort, though diligently looked for. The proportion of albumen was no more than corresponded to the blood present.

The patient from this time gradually but steadily improved. The bladder slowly recovered the power to expel its contents; the urine became less and less bloody, and finally clear, and free from albumen.

Eight months afterwards (August 26, 1862) the patient waited on me. He was still pale and thin, but reported himself well, and had for the last six months been able to pursue his avocation.

I heard nothing more of the case until June 22, 1863, when I was summoned to visit the same man with Dr. Nesfield, under whose care the patient came after Dr. Crompton's departure from town. I found him in a desperate condition—emaciated to a skeleton; so weak that he could not turn in bed, nor raise his head from the pillow. There was no anasarca nor ascites. Great pain was complained of in the right renal region, but no tumor or fulness existed there. The urine was loaded with pus, and highly ammoniacal. Six days after, the patient died.

Autopsy.—On opening the abdomen and pushing aside the small intestines, a cancerous mass, half as large as the fist, was found implicating the base of the bladder, especially about the entrance of the right ureter. Within the viscus, a soft sprouting fungus of the size of a hen's egg, was seen springing from the trigone; it was rounded in shape, elevated about an inch above the level of the mucous membrane, and very red. On and about it, occupying the inequalities of its surface, lay a quantity of calcareous or phosphatic matter, deposited in irregular masses. Small masses of a similar nature had been observed to come away with the urine for some weeks before death.

The right kidney was a little larger than natural; it felt flaccid and hollowed. On section, six cancerous nodules as large as marbles, and several smaller ones, were counted in the cortical substance. None of these were softened, nor communicated in any way with the pelvis of the kidney. The organ was sacculated to a considerable extent. The pyramids were in great part absorbed, and the remainder of the renal structure was converted into a reddish, leathery substance. The pelvis and infundibula were much dilated. The ureter was enlarged to the size of the index finger, and near its entrance into the bladder, its calibre was almost effaced by the cancerous mass at the base of the bladder. through which it passed. Broken fragments of calcareous matter lay scattered in the dilated pelvis, which, together with the ureter, contained a quantity of urinous ammoniacal pus.

The left kidney contained eight or ten nodules similar to those in the right. The intervening renal tissue was perfectly healthy; the ureter was free, and the pelvis undilated.

This case presented several points of difficulty. At the time of my first visit the symptoms indicated pretty clearly an affection of the bladder: and as no stone could be detected on sounding, and no pus passed with the urine, the probability of the existence of a bleeding fungus seemed strong. The other possibility was renal calculus. The previous history favored the latter view; the patient had recovered perfectly from his first attack of hæmaturia three years before—a result quite conformable with the idea of renal calculus, but much less so with that of fungus of the bladder. Then again, how explain the ascites and anasarca? They could not be attributed to the losses of blood and hydræmia consequent thereupon, for they passed away before the hæmaturia ceased. It appeared more likely, that the dropsical symptoms and the paresis of the bladder were companion phenomena, of a paralytic nature, produced by the reflex results of the antecedent intense irritability of the bladder, acting upon the nerves of the bladder and of the bloodvessels of the lower half of the body.

APPENDIX.

SARCOMA OF THE KIDNEY.—(*See* Robson, "Brit. Med. Journ.," 1876, I. p. 232; Baginski, "Deutsch. Med. Wochenschr.," 1876, No. 10; Geddings, "Trans. Americ. Gynæc. Society," 1877, p. 479; Whitehead and Dreschfeld, "Brit. Med. Journ.," 1881, II. p. 741; Fotherby, "Brit. Med. Journ.," 1882, I. p. 157; Heath, "Brit. Med. Journ.," 1882, II. p. 100; Abercrombie, "Path. Trans.," xxxi. p. 168; Day and Thornton, "Path. Trans.," xxxii. p. 142; also *see* Virch. and Hirsch., "Jahresber.," 1880, vol. ii. p. 212; and Cornil and Ranvier, "Histologie Pathologique," 2d edition, 1884, vol. ii. p. 638.)

Many of the cases mentioned above under the head of Cancer of the Kidney were observed before the differentiation between carcinoma and sarcoma had been generally demonstrated. It is probable that a considerable number of the older cases would now be classed with the latter variety of tumor. In those cases in which the sarcomatous nature of the growth has been recognized, the tumor has usually been found to be composed of small round cells, or of these mixed with spindle cells. Lymphosarcoma of the kidney is by no means rare, especially in young subjects.

Clinically no sign has yet been observed, which will serve to distinguish sarcoma from carcinoma of the kidney. The clinical features of such growths may be illustrated by the following account of a case under the care of my colleagues, Dr. Dreschfeld and Mr. Walter Whitehead.

A. L., aged 46, came to the out-patients' department of the Manchester Royal Infirmary for the first time on April 4, 1882, complaining of the occasional passage of blood in his urine. He stated that he had always enjoyed good health; he had suffered from gonorrhœa, but not from syphilis. He was a well-built but spare man of very dark complexion; he had a slightly atheromatous pulse and beginning arcus senilis. On the right side of the abdomen a small firm, freely movable globular tumor could be felt; its upper border was about one inch below and separate from the liver; its lower border was in a line with and about two inches to the right of the umbilicus. Percussion gave a dull sound, and no bowel could be detected over the tumor. It was perfectly painless. The patient had been aware of its presence for some time, but felt no inconvenience from it. Percussion of the lumbar regions behind gave a duller sound on the right than on the left side, but no fulness could be detected on that side. The urine was uniformly dark red, containing a considerable amount of blood intimately mixed with it. Microscopically examined, it showed, besides blood-corpuscles, some large round cells with large nuclei, which filled up nearly the whole of the cell. There were no renal casts. The remaining organs were normal. A diagnosis was made of tumor of the right kidney, probably of sarcomatous nature.

In the further progress of the case the tumor increased in size. It still remained painless, and extended downwards and to the side, so that it could be easily grasped by one hand being applied to the right lumbar region behind and the other over the tumor in front; it could thus be moved both laterally and vertically; the surface of the tumor felt smooth and inelastic.

At the beginning of August the patient complained of feeling weaker, and of suffering from flatulence and occasional vomiting. The case being one where an operation was indicated, Mr. Whitehead removed the growth on September 5th, but the patient died on September 9th. The growth proved to be a large round-celled sarcoma of the kidney, and at the autopsy no secondary growths were found.

A sarcoma in which striated muscular fibres are found, occurs very rarely and always in young children. Specimens of such a tumor were exhibited at a meeting of the Pathological Society on Nov. 1, 1881, by Mr. Eve and Dr. Dawson Williams. The explanation offered by Cohnheim of the presence of muscular fibres in these tumors, is that, owing to a faulty segmentation of the protovertebræ, some of the germinal muscle-cells are mixed from the first with the rudiments of the uro-genital organs and these germinal cells afterwards develop into a pathological new growth. The Committee of the Pathological Society considered that the tumors mentioned above were developed from the remains of the Wolffian body. Only twelve such cases have been as yet described, and of these the following is a list:

Eberth—Virch. Arch., vol. 55, p. 518.
Cohnheim—Ibid., vol, 65, p. 64.
Brodowski—Ibid., vol. 67, p. 205.
Marchand—Ibid., vol. 73, p. 289.
Brosin—Ibid., vol. 96, p. 453.
Kocher and Langhans—Deutsche. Zeitsch. f. Chirurg., Bd. ix.
Huber—Deutsches Arch. f. klin. Medicin., vol. xxiii. p. 312.
Landsberger—Berl. klin. Wochensch., 1877, p. 497.
Osler (2 cases)—Journal of Anat. and Physiol., vol. 14, p. 229.
Eve—Path Trans., vol. xxxi. p. 164.
Williams—Ibid.

CHAPTER XI.

BENIGN GROWTHS IN THE KIDNEY.

In the records of medicine a number of cases may be found, in which the kidneys were the seat of adventitious growths of osseous, fibrous, fibro-fatty, cartilaginous, or glandular tissue. Generally speaking, such growths do not, unless they are large enough to constitute a palpable tumor in the abdomen, produce any appreciable symptoms during life; and they offer more of a pathological than clinical interest. They are all extremely rare.

1. Osseous Growths.—Mention has already been made of the ossification which sometimes takes place in the fibrous septa which separate the compartments of a sacculated kidney (*see* p. 456).

Sometimes a fibrous or cartilaginous tumor grows in the substance of the kidney, and subsequently ossifies, transforming a large part of the organ into a bony mass. The tunica propria has also been known to undergo ossification. Rayer states that Dr. Elliotson sent to him two bony shells formed by the ossified tunica propria and pelvis of the kidney, taken from a man who died with symptoms of apoplexy.

2. Fibrous and Fibro-fatty Growths.—Dickinson and Bristowe have each recorded a case, in which the major part of the kidney was replaced by a morbid growth, composed of a matrix of fibrous tissue, in the interstices of which were soft masses of free fatty matter unenclosed in cells. In Dickinson's case the tumor weighed 6 lb. 7½ oz., and formed a perceptible tumor in the right hypochondrium. After death, a coil of intestine was found in front of the tumor, but so compressed and empty that its nature was not likely to be recognized during life. Wilks records a case in which a tumor was found on the right side of the abdomen six years before death; it was partly solid and partly fluid, and was by some diagnosed as cancerous. On post-mortem examination, the right kidney was found converted into a tumor the size of a young child's head. This was found to consist of a solid growth on one side bearing a striking resemblance to the fibro-cartilaginous tumors sometimes met with in the neck, but which was found, on microscopic examination, to consist exclusively of fibrous tissue; and on the other side a cyst was found, which on section was seen to be due to the enormously distended pelvis. The new growth had slowly invaded and

destroyed the renal tissue, while it gradually distended the capsule, and thus preserved the general form of the kidney.

In the case described by Godard, the lower half of the kidney was converted into a large mass of ordinary adipose tissue. A calculus of considerable size was lodged in the dilated pelvis. A somewhat similar transformation is described by Dr. Hullett Browne, complicated with calculous pyelitis, and renal fistula opening in the left loin ("Path. Soc. Trans.," xiii. 132).

Adipose tissue is, in other cases, deposited in great quantity, not in, but around the kidneys, so as evidently to interfere with their functions. In the museum of the Manchester School of Medicine there is a preparation in which a pale and atrophied kidney is enveloped in a firm investment of dense, granular, fibro-fatty tissue, fully an inch thick. The same tissue penetrates deeply into the hilus, so as to compress the bloodvessels and excretory channels.

3. LYMPHATIC GROWTHS.—Virchow, Friedreich, and Böttcher have described growths or deposits in the kidneys of leucocythæmic individuals, similar to those found under the same circumstances in the spleen and lymphatic glands.

4. SYPHILITIC DEPOSITS IN THE KIDNEYS.—It seems well ascertained that the waxy or lardaceous type of chronic Bright's disease is frequently due to constitutional syphilis. Out of 145 cases, collected by Fehr, 34 were attributed to this cause (*see* p. 406); and in 27 cases of constitutional syphilis examined by Dr. Moxon,[1] the kidneys were found lardaceous in no less than 12 instances.

The occurrence of gummy tumors and cicatrices (such as occur in the liver) is rare, though not unknown, in the kidneys of syphilitic persons. Both Cornil and Lancereaux give examples. In Lancereaux's case there were found on the surface and in the thickness itself of the cortical substance of the kidneys small tumors of the size of a pea, of a yellowish-white color, and presenting, on examination by the microscope, the cellular and nucleated elements found in gummy syphilitic tumors elsewhere.[2]

Dr Moxon gives a remarkable case, in which a gummy tumor, as large as a small potato, existed in the left kidney of a syphilitic woman. It had a regular nodose outline, and was composed of a yellowish substance, quite uniform in appearance, and which was firm, hard, and tough. It yielded no juice on scraping. Under the microscope, the mass was found to consist of small corpuscles crowded together, first obscuring, and then destroying and replacing the proper tissue, and then themselves perishing into a heap of fat grains and globules—all which exactly corresponds to the usual character of syphilitic gummata. The general character of the kidney was that of the large pale lardaceous kidney.[1]

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[1] A Contribution to the History of Visceral Syphilis, by Dr. Moxon. Guy's Hosp. Rep., 1868.

[2] Lancereaux. Treatise on Syphilis—Syd. Soc. Trans., vol. i. p. 298. A case of syphilitic gummy tumors of the kidney is recorded at great length (with a drawing) by Paolucci from the Clinique of Prof. Cantani, of Naples, in Il Morgagni for June, 1874, p. 413. *See* also Professor Greenfield, Résumé of Renal Pathology in the Syden. Society's Atlas.

5. Wagner has published two cases in which one kidney was converted into a large tumor, composed apparently of a combination of epithelial structure, fibrous tissue, and glandular (pancreatic) sarcoma. Both were female children—one nine months and the other eight years old.

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[1] The power of the syphilitic poison to produce acute Bright's disease appears doubtful. Lancereaux cites two cases of albuminuria reported by Perroud, which accompanied the secondary period of syphilis—some four or six months after the indurated chancre—and which appear to have issued favorably. The late Mr. Bradley, of this town (Brit. Med. Journ., 1871, i. p. 116), has also recorded a case of acute Bright's disease, with general anasarca in an infant suffering from congenital syphilis. The renal affection subsided *pari passu* with the disappearance of the cutaneous syphilis. Until further proof, it may be doubted whether these were not examples of a fortuitous coincidence of two independent morbid conditions.

CHAPTER XII.

TUBERCLE OF THE KIDNEY.

Deposits of tubercle in the kidney may be *primary* or *secondary*. In the former case the kidney and its appendages are the seat of extensive disease, which runs on, attended with severe urinary symptoms, generally, if not always, to a fatal conclusion. In the latter, the deposits form as a part-manifestation of general tuberculosis, or constitute incidents in the course of primary tubercle of the lungs, intestines, or some other organ; secondary deposits rarely give rise to symptoms, and are mostly unsuspected until the autopsy.

The comparative frequency of tubercle in the kidney may be judged of by the following numbers, which must be understood to embrace both primary and secondary deposits—the latter being, especially in children, by far the most frequent. Out of 1317 tuberculous subjects, examined in the Pathological Institution of Prague (out of a total of 6000 bodies), tubercle in the kidneys was found 74 times, or in the proportion of 5.6 per cent. of all tuberculous subjects.[1] Among 315 tuberculous children, Rilliet and Barthez found tubercle in the kidneys 49 times, or in the proportion of 15.7 per cent. From these statistics we may gather that the kidney is nearly three times more liable to deposits in tuberculous children than in tuberculous adults.[2]

A.—PRIMARY TUBERCLE OF THE KIDNEYS.

(Tuberculous Pyelitis.)

The statements made in the following pages are mainly based on an analysis of 35 cases, derived from various sources.

Morbid Anatomy.—The disease (which always implicates more or less extensively the excretory apparatus as well as the gland itself) begins in the kidney, and extends downwards into the pelvis, ureter, and bladder; or it begins in the pelvis, and spreads upwards into the kidney, and downwards towards the bladder; or all these parts may be invaded simultaneously or in quick succession. In the kidney, the deposit begins in the form of gray or yellow nodules in the cortical part: these afterwards coalesce into larger masses of crude tubercle, and extend into the pyramids. These masses at length soften in the centre, and eventually open into the infundibula. In this way abscess-like cavities arise, with anfractuous boundaries of tuberculous matter, which communicate with the pelvis, and discharge pus and broken masses of tubercle into the stream of urine.

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[1] Prager Vierteljarsch , Bd. 1. S. 1 (1856).

[2] I omit the statistics of Dr. Chambers, because there are some discrepancies in his tables which I have been unable to reconcile.

In the pelvis and ureter, the deposit first begins in the submucous cellular tissue,[1] where it forms a rough, granular, semi-transparent or opaque layer. It consequently softens and disintegrates, causing extensive destruction of the superjacent mucous membrane, which is discharged in shreds with the urine, mixed with pus and blood. The deposit is sometimes so abundant and uniform in the ureter, that that tube is converted into a thick rigid cylinder, of which the available bore is greatly narrowed, or even altogether obliterated. In a specimen submitted to me by Dr. Leech (case to be presently related), the interior of the pelvis was thickly encrusted with calcareous matter, and one of the ureters was completely occluded near its centre by an oval mass of tubercle about the size of a horse-bean.

Extensive destruction of the renal tissue eventually takes place, both from the encroachment of the tubercle masses, and from sacculation and dilation of the organ by the blocking up of the ureter (pyonephrosis). Sometimes no vestige of the secreting tissue remains; but more commonly certain portions are preserved, and these may present a moderately healthy appearance, or be far advanced in degeneration. In other cases the ureters are open and dilated, and admit free passage to the urine, pus, and tubercular débris; the kidney then maintains its normal dimensions, or it may even be contracted.

Actual tumor (pyonephrosis), detectable during life, is mentioned in 7 out of our 35 cases. It seldom reached great dimensions, but in one instance related by Ammon, it filled the entire side of the abdomen, from the false ribs to the crest of the ilium.

The disease is sometimes limited to one side, but much more frequently it invades both. Out of 32 cases which supply information on this point, the two sides were affected in 19, and one side alone in 13 cases. Of the latter, the right kidney was affected 7 times and the left 6 times.

In addition to the kidney itself, and its immediate appendages (pelvis and infundibula), the disease almost invariably involved the ureter (in 30 out of 32 cases), and very frequently the bladder (in 21 cases). The urethra was involved in 7 cases.

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[1] *See* an observation by Dr. Handfield Jones, in the first vol. of the Path. Soc. Trans., p. 283.

In the male sex, the disease not unfrequently implicates the generative organs (prostate nine times, vesiculæ seminales six times, testicles four times); but it is otherwise in the female sex. Out of nine females, in only one instance (to be presently related) were any of the generative organs involved.[1]

The disease very rarely runs its entire course without the occurrence of tuberculous deposits in other and unconnected parts of the body. Thirty cases were examined with sufficient minuteness to supply information on this point. The lungs were affected 28 times; the abdominal glands, 14 times; the intestines, 19 times; the osseous system, 5 times; the peritoneum, 5 times; the spleen, 3 times; and the liver, once.

In one case the ulceration (tuberculous) in the bladder opened a communication with the rectum (Basham); in another a vesico-vaginal fistula resulted from a similar cause (Mosler); in a third, the suppurated kidney burst into the peritoneal sac (Lundberg, Schmidt's "Jahrb.," Bd. xci. S. 74).

Etiology.—The direct exciting cause of renal tubercle is generally inscrutable. Cold is the cause most frequently mentioned; the patients came, in several instances, from conspicuously tuberculous families. Men are more liable to this complaint than women—in the proportion of 21 of the former to 12 of the latter. No age is altogether exempt. The youngest case noted was a child of three years and a half, and the oldest (mentioned by Dittrich, and not included in the table) was a man of seventy-one; but the greater number occurred in the middle periods of life. The following table gives the precise ages in 31 cases:

From	0 to 10 years	4 cases.
"	10 to 20 "	5 "
"	20 to 30 "	6 "
"	30 to 40 "	9 "
"	40 to 50 "	5 "
"	50 to 60 "	2 "

Symptoms.—The symptoms are mainly those of chronic pyelitis, conjoined, in a considerable majority of the cases, with those of chronic cystitis. The complaint begins with a dull pain in one or both lumbar regions, accompanied with frequent micturition. At the same time the urine becomes turbid, and sometimes mixed with blood. When the disease is fully established, the urine is charged with a large quantity of pus, which forms a thick, yellowish layer at the bottom of the vessel.[1] Blood is also usually present, either in microscopic quantity, or sufficiently to tinge the urine. The hæmaturia is, however, never profuse; in several instances it was noted that small, thready clots of blood were passed. Under the microscope, there are found, in addition to the pus and blood-corpuscles, a number of oval and irregularly tailed cells from the bladder and upper urinary passages, together with granular detritus, broken masses of softened tubercle, shreds of connective tissue and elastic fibres.

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[1] The mutual independence of tuberculosis of the urinary and generative systems in the female, is further shown in a converse manner by Dittrich. Out of 45 cases of tuberculosis of the female genital organs, he only found one in which the disease also implicated the urinary organs (Archiv der Heilkunde, 1863, p. 304). Virchow describes an additional example of this rare conjunction, in which urinary tuberculosis was associated with secondary deposits in the vagina (Archiv für Path. Anat., Bd. v. S. 405).

The reaction of the urine is feebly acid. Very few exceptions to this rule exist, and those are due to ammoniacal decomposition of the urine from detention in some part of its course, as in Mosler's case, from the tumid state of the external genitals. The urine is necessarily albuminous from the presence of pus, but usually only in a slight degree. Casts of tubes are only mentioned once. Micturition is always excessively frequent; often dolorous. In two cases, temporary alleviation of the pains followed each micturition: this was not observed in other cases.

As the disease advances, great emaciation takes place, accompanied with hectic fever, sometimes marked by chills and rigors of tolerably regular recurrence. Persistent pains are felt in the back, in the lower part of the abdomen, and often along the urethra.

When the kidney is sacculated and enlarged, so as to form a tumor in the flank, the swelling is usually painful; it may, or may not, yield distinct fluctuation. Sometimes the tumor displays variations in its size: it enlarges when the ureter is dammed-up by the discharged débris, and becomes more painful, at the same time the quantity of pus in the urine diminishes—or, if the stoppage be complete, temporarily disappears. Anon the course of the pus and urine is reëstablished, and the tumor subsides and becomes less painful.

In the progress of the case, or towards its termination, the lungs and intestines generally betray the advance of tuberculous disease. Cough and oppression of the chest, or uncontrollable diarrhœa, make their appearance. Gastric symptoms (nausea, vomiting, hiccough) are unusual; but in some cases, as in the two about to be related, they are a marked feature of the complaint. The absence of intestinal tuberculosis, accompanied with obstinate constipation, appears to favor their occurrence.

If both kidneys are affected, the extensive destruction of secreting tissue is liable to give rise to uræmic phenomena. The quantity of the urine is usually below the average; but exceptionally, as in a case recorded by Sir Risdon Bennett ("Path. Soc. Trans.," viii. p. 284), the urine is abundant and of low specific gravity. Usually death occurs from the exhaustive effects of the protracted and profuse suppuration, or from the severity of the pulmonary or intestinal complications.

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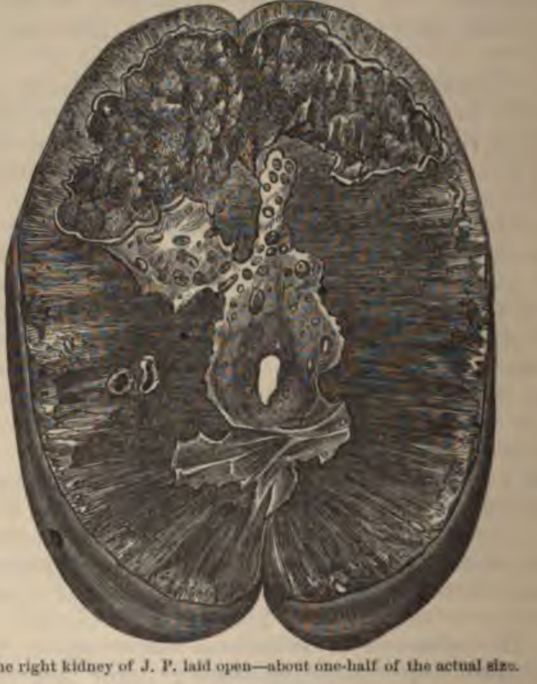
[1] At intervals the urine may be copious and limpid, containing no albumen, and giving no deposit. (Tapret.)

The following cases will serve as illustrations of the course of the disorder and of the appearances generally found after death.

CASE 1. *Tuberculous disease of the right kidney and ureter, and of the bladder, urethra, and prostate; absence of left kidney and ureter.*—J. P., a packing-case maker, æt. 23, was admitted into the Manchester Royal Infirmary, under my care, March 27, 1871. He was suffering from old-standing discharge of large quantities of pus with the urine, mixed with a little blood. The disease had existed nine months; the emaciation was extreme. There were distinct signs of consolidation in the apices of both lungs, but no cough or expectoration. Profuse night-sweats and an elevated temperature in the evenings bore evidence of hectic fever. The tongue was dry and red; the lower part of the belly was the seat

FIG. 68.



The right kidney of J. P. laid open—about one-half of the actual size.

of a dull aching, and micturition was both frequent and painful. He sank from exhaustion a few days after his admission into hospital.

Autopsy (30 hours after death).—*Pleura* universally adherent on right side; no adhesions on left side. There was a large amount of gray tubercle in the upper lobes of both *lungs*, the lower lobes being free; the tubercle was associated with a quantity of pigmentary deposit. No cavities or purulent exudation observed. *Heart* and *pericardium* healthy. *Liver* enlarged, soft, and friable, presenting a pale surface on section; *spleen* healthy. The *left kidney, suprarenal capsule*, and *ureter* were absent. The *right kidney* was considerably enlarged, and weighed 14½ ounces. On section, there was a large cavity at the upper part, occupying one-fifth of the entire kidney, filled with pus, and two other very much smaller purulent cavities in the central part of the organ (*see* Fig. 68). The cortical portion was seen to be much hypertrophied, but pale, except the parts situated between the pyramids, which were dotted over with red points; the pyramidal portion was atrophied and almost entirely absent. The pelvis was dilated and divided into pouch-like enlargements, the lining membrane being studded over with small whitish deposits of tubercular matter, which could be traced along the ureter to the bladder, and thence along the urethra. About half an ounce of pus escaped from the ureter on section. The ureter was dilated at its commencement and near its termination, where it easily admitted the forefinger; but the opening into the bladder was so small that it could not be detected until a probe had been passed from without. A slight depression marked the position for the entrance of the left ureter, of which, however, no trace could be found. The fundus of the *bladder* was covered with tubercular deposit, which was limited to this part, the body and summit being entirely free. The mucous membrane of the affected part was much broken down in structure, and in parts almost ulcerated through; the walls of the bladder and ureter were considerably thickened. The *spermatic cords* and *vesiculæ seminales* were normal, being perfectly free from tubercle. The *prostate* was involved in the disease, and partially disintegrated.

CASE 2. *Pericarditis, with subsequent adhesion; cirrhosis of liver, and enlarged spleen, followed eight years after by tubercular pyelitis and general tuberculosis.*—I first saw G. P., a grocer, æt. 27, in 1860. He was then suffering from plastic pericarditis, with immense enlargement of the spleen and considerable enlargement of the liver. His illness had been brought about by intemperance. He was in the habit of taking great quantities—as much as a quart a day—of gin. From this illness he slowly recovered, at least so far as to be able to go about and look after his business; but the enlargement of the spleen remained. I saw this man occasionally until 1867. He persisted, with some intermissions, in his intemperate habits, and failed in his business. In August, 1867, he appeared among my out-patients at the Infirmary. The spleen was now decidedly smaller, and the heart appeared quite healthy; but he was complaining of frequent micturition and of passing blood with the urine. The blood first appeared in the previous June, and had continued ever since. In November, 1867, I admitted him as an in-patient into the Infirmary. His condition was then as follows: he was somewhat emaciated, skin moist, finger-ends slightly clubbed, no œdema anywhere, pulse 96, respiration 20, tongue dry and fiery-red. The abdomen was rather tumid; the liver extended from the nipple to two inches below the costal margin; the spleen measured nine inches vertically. The urine was loaded with pus, and with enough blood to give it a full red tinge. The proportion of blood to pus in this case was much greater than is usually the case in tubercular pyelitis. There were no casts in the urine, and the albumen did not exceed the proportion due to the admixture of pus and blood. The patient remained in the Infirmary fourteen days, and underwent little or no change. He did not keep his bed. Pulse varied from 70 to 80, respiration 20. He continued to pass rather more pus and rather less blood; but there was always enough of the latter to color the urine, and often small clots. The tongue retained the same "broiled ham" appearance. It is to be remarked that the spleen was now much smaller and the liver larger than when I first saw him seven years before.

He was again made an out-patient, and I saw him from time to time. He continued without much change, passing purulent and bloody urine at very frequent intervals, until the end of January, 1868, when he was seized with uncontrollable vomiting, under which he sank on February 2d.

Autopsy.—Emaciation only moderate; no œdema. The liver was cirrhotic in an advanced degree, the spleen enlarged; but both organs (especially the spleen) were much smaller than they had been eight years ago. The lungs were thickly studded with gray granulations from base to apex. The pericardium was adherent throughout, but it was not thickened, and the heart itself was healthy. Both kidneys were deeply affected with tuberculous pyelitis. The pelves were much enlarged, and studded with broken-down, ulcerated tubercle. A few masses of yellow, unsoftened tubercle were found in the right kidney, about the bases of the pyramids. The pyramidal portions were in great part destroyed by the encroachment of the distended pelves. The ureters were lined throughout with ulcerated tubercle; the bladder and urethra also were partially affected in a similar manner. The peritoneum was adherent almost throughout, and contained here and there tuberculous granulations.

The next case occurred in the practice of my colleague, Dr. Leech, who kindly furnished me with the notes of the case, and with the anatomical preparations.

CASE 3. *Tubercle in both kidneys, ureters, bladder, and urethra; in the prostate gland and vesiculæ seminales; also in the lungs and mesenteric glands.*—W. P., æt. 53, a brewer, had been ailing three years. His disease began with pain and difficulty in micturition. The urine was thick, and sometimes mixed with blood; though he made water very frequently, he did not think that he passed an excessive quantity. Except for short intervals, he had suffered from the same symptoms for the last three years. His urine had been occasionally quite clear, but generally thick, and often dark. He had never complained of much pain in the lumbar region.

About nine months before his death, the patient began to vomit frequently, especially after taking food; for the last five months vomiting after meals had been constant, frequently accompanied with pain in the epigastrium.

The general health had gradually failed during the last three years; but he lost flesh and strength more rapidly during the last twelve months. He worked occasionally, however, up to six months before his death. For many months he had felt a gnawing pain just over the pubes, increased by pressure; this was less severe during the last six months of life.

Five months before his death the edges of the meatus urinarius began to ulcerate, and the ulceration gradually widened the orifice to double its natural size. In the course of the last six months two small abscesses formed in the scrotum, both of which were opened, and subsequently healed.

He had been in the habit of taking large quantities of beer, but not much spirits; he contracted gonorrhœa many years ago, but he never had any venereal sores.

About a week before his death, he was in the following condition: Emaciation very great; countenance sallow; meatus urinarius much enlarged and ulcerated; severe pain is felt along the urethra and in the glans penis, especially after voiding urine. The ulceration can be seen to extend for a depth of nearly half an inch into the urethra. The urine contains abundance of pus and a small quantity of albumen; no casts were found. The urine is passed very frequently, and in small quantities. He vomits after everything he takes, even after simple water, or a little brandy and water. What he brings up is a brownish liquid; it contains no sarcinæ; sometimes a little blood comes up; but he thinks it is derived from the back part of the nose, where he feels pain and rawness. The abdomen is flat, or rather depressed; the epigastrium is very painful on pressure. In the right hypochondrium a little hard mass can be felt on deep palpation, and there is dulness at this spot on deep percussion. There is also considerable pain on pressure in the hypogastric region. He complains of aching pain over the lower ribs on both sides. The bowels are very constipated, and have been so for some time.

The day before his death he vomited a considerable quantity of blood. The sickness and vomiting were somewhat relieved for a short time by effervescing draughts, with morphia, but only for a day or two. Afterwards the vomiting became continuous; he vomited, or attempted to vomit, every half hour or so. There was no delirium till the day before his death, which took place on the 27th of December, 1864.

Autopsy.—Stomach of normal size; mucous membrane congested in parts; no thickening of, or deposit in, the walls; pyloric valve thickened and somewhat contracted. *Liver* healthy. *Mesenteric glands* much enlarged; some of them contained small cretaceous masses. *Lungs: left* contracted and full of miliary tubercles; *right* contained hard masses of tubercle at apex.

Kidneys: left of natural size; on the outside, white, slightly raised spots are seen through the fibrous covering. On removing the latter, the surface of the cortex is seen marked with small white nodules, some of which are collected into patches; to these patches the tunica propria is tightly adherent. On section, several large cavities are opened into, containing pus. The largest of these is situate in the upper part of the kidney, and is lined by a smooth membrane, except at its opening into the pelvis, where some calcareous matter is deposited. All the other cavities open into the pelvis; some of them have irregular anfractuous boundaries of softening tubercle; these likewise are more or less completely lined with calcareous matter, composed of carbonate and phosphate of lime. The whole of the pelvis is encrusted with the same earthy material, which can also be followed for some distance down the ureter. In the cortical and pyramidal parts of the kidney intervening between the cavities, the renal tissue is studded with soft nodules of disintegrating tubercle, varying in size from a pin's head to a pea. The submucous tissue of the pelvis and ureter is the seat of a thick granular layer of gray tuberculous matter, softened in parts; the ureter is thereby converted into a thick, rigid, uneven tube, with a narrowed calibre.

The *right kidney* is much smaller than the left. The pyramids are occupied by abscess-like cavities full of pus. The septa between the pyramids are in some places preserved, in others partially broken down. The pelvis is greatly contracted, almost obliterated; in one or two places there are narrow communications between the sacs of pus in the pyramids and the unobliterated parts of the pelvis. The ureter is completely occluded, midway between the kidney and the bladder, by an oval nodule of yellow crude tubercle about the size of a horse-bean.

The whole of the mucous membrane of the *bladder* is strewed with deposits of tubercle. These are sparsely scattered and scanty, except over the trigone; here the deposit is very abundant, in the form of small granulations rather larger than a pin's head. A few similar granulations are seen in the prostatic part of the urethra.

The *prostate* gland is somewhat enlarged on the under surface, and contains two small tuberculous nodules. One of the *vesiculæ seminales* also contained soft tuberculous matter.

CASE 4. *Tubercle of the left kidney, pelvis, and ureter; of the bladder and urethra; also of the pericardium, lungs, peritoneum, and mesenteric glands.*—A needlewoman, 33 years of age, strongly built, whose father seems to have died of phthisis, took cold in consequence of a severe wetting in the autumn of 1859, about a year before her death. Her first symptoms were those of cystitis, with moderate fever. The fever soon disappeared; but the pain in the bladder, which radiated upward into the left loin, and the urgency and burning pain of micturition, together with a turbid condition of the urine, remained, and persisted through the winter. Impairment of digestion and emaciation were also observed.

About half a year from the commencement of her complaint, feverishness returned; pain and urgency of micturition increased, and blood appeared in the urine. From this time (February, 1860) the patient became the object of exact observation. She was already markedly emaciated, pale with a hectic flush on each cheek; she suffered from headache, often from palpitation; the appetite was bad, the bowels confined, and there was moderate fever; the desire to pass water was constant, and the pain in the bladder, shooting into the left loin, great. The urine was scanty, tinged with blood, with a thick deposit of pus and blood-clots. The urethra was somewhat swollen and tender; and after micturition the bladder still contained several ounces of unevacuated urine. These symptoms maintained themselves without essential change for six months, until she died on the 4th of September.

The loss of flesh continued without interruption, and reached an extreme degree. Hectic fever prevailed, with evening exacerbations—the temperature rising to 38.5°–39.5° C., and sometimes to 40° C. From the end of March, severe paroxysms of chills and rigors, followed by heat and sweating, occurred at irregular intervals. At the end of July, night sweats and bed-sores were noted.

Gastric symptoms were throughout prominent. They increased and diminished. For days together the patient would suffer from severe epigastric pains, nausea, vomiting, and disgust of food. From the end of May, she occasionally suffered from paroxysms of hiccough, lasting several hours. In the last moments of life she was troubled with bilious vomiting, and towards the end she had diarrhœa.

The quantity of urine was invariably scanty, though the patient sometimes drank a good deal. The proportion of blood in the urine gradually diminished from the beginning of March, and the blood-clots sometimes were absent for several days, yet the blood never disappeared altogether. The quantity of pus increased. From May onward there appeared occasionally in the urine sloughy shreds of cellular tissue, elastic fibres, swollen bladder-epithelium, and little yellow broken masses of detritus. On the 8th of July, some epithelial renal casts were for the first time discovered in the urine.

At the end of April a vaginal examination revealed the existence of a small hard swelling at the base of the bladder (this was proved at the autopsy to be due to a tuberculous thickening at the point of entrance of the left ureter).

The patient began to complain of pains in the chest soon after her admission into hospital; then a dry cough came on, and later on, a slight dulness on percussion was perceived in the left infra-clavicular region. In the later periods she also complained of oppression in the chest; but she never expectorated, and there never existed any of the more open symptoms of pulmonary tuberculosis.

It was a singular and inexplicable circumstance, that a short time before death an improvement took place, which lasted several days; all the pains and the fever disappeared, the appetite returned, and the strength was so far restored that she was able, unassisted, to sit up, although before she was scarcely able to turn in bed.

Autopsy.—Emaciation had reached the most extreme degree. The *brain and its membranes* were healthy. Some tuberculous granulations were found in the otherwise healthy *pericardium* at the base of the right auricle. *Heart* healthy. The *lungs* and *pleuræ* were studded with gray and yellow granulations. The left apex contained several gray nodules as large as walnuts. The *liver* contained no tubercle, but its peritoneal investment was thickly covered with gray granulations; the organ was adherent, by its convex surface, to the abdominal wall. A mass of tuberculous glands, as large as a pigeon's egg, occupied the portal fissure.

The *peritoneum* covering the spleen, intestines, and mesentery was thickly covered with tuberculous granulations. The *mucous membrane of the intestinal canal* was throughout free from tubercle.

The *right kidney* was itself healthy, together with its pelvis and ureter; but its capsule was studded with miliary tubercles.

The *left kidney* presented its usual form and size, but its capsule was converted into a thick membranous covering which enclosed the degenerated gland. In the upper half of the organ were found one large and two smaller cavities, separated incompletely from each other by undestroyed renal tissue. These cavities were filled with a greenish-yellow muco purulent fluid, and their anfractuous walls were composed of reddish and yellow cheesy deposit. The reddish and yellow materials, in the form of smaller and larger nodules, were deposited in close contact with each other. These cavities stood in direct continuation with the mucous membrane of the pelvis, which was similarly degenerated and thickened. In the lower half of the organ was another and a larger cavity, similarly constituted with the others, and, like them, communicating with the pelvis.

The undestroyed portions of the kidney had a pale red color, and contained in the lower parts of the gland several small, roundish, grayish, and yellowish cheesy nodules. The coats of the left ureter were several lines thick, firm, and rigid; the mucous membrane tumid, friable, yellowish, and the seat of numberless miliary tubercles; in many places the ureter was superficially eroded into roundish ulcers, with firm raised edges.

The walls of the *bladder* were three lines thick; its cavity was contracted; its inner surface reddened, and riddled with ulcers having tuberculous margins. The peritoneal surface of the viscus was studded with tubercles. The serous coverings of the generative organs were in the same condition, but the organs themselves, with the exception of the ovaries, were healthy. The ovaries contained several gray tuberculous nodules.

The lumbar glands contained cheesy matter. (Kussmaul, "Würzb. Med. Zeitsch.," Bd. iv. S. 24.)

THE DURATION of the disease varies from a few months to two or even three years. Only 14 out of our 35 cases supply moderately exact information on this point. Five died under six months; five in six to twelve months; three in one to two years; and one survived three years.

THE DIAGNOSIS of tubercle in the kidney and its appendages turns mainly on the existence of signs of chronic pyelitis, joined with collateral evidence of tuberculosis, and the absence of any other assignable cause of pyelitis (calculi, hydatids, etc.). Examination of the urine furnishes important information; not only is the urine abundantly purulent, but it also contains a quantity of granular débris, sometimes mixed with broken masses of tuberculous matter (insoluble in acetic acid), shreds of connective tissue, and beautiful meshes of elastic fibres from the cast-off patches of disintegrated mucous membrane. During recent years the tubercle-bacillus has been discovered in such urine by Rosenstein,[1] and also by Babés.[2] Its detection adds great support to the diagnosis.[3] The severity of the general symptoms—the progressive and great emaciation and failure of strength—must also be taken into account. When evidence of pulmonary phthisis, or ulceration of the bowels exist, they supply a valuable indication; but it should not be forgotten that, although tubercles almost invariably exist in these cases in the lungs or intestines, they often run a latent course, or are not in a sufficiently advanced stage to be clinically detected.

From cancerous pyelitis (without tumor) the diagnosis is generally established without difficulty by the characters of the urine. In cancer, the urine (if not normal) is bloody rather than purulent; in tubercle, it is always immensely purulent, and only slightly, or not at all, bloody.

It need scarcely be stated that primary tubercle of the kidney is not capable of diagnosis until it has softened and commenced to be discharged.

THE PROGNOSIS is excessively grave, if not absolutely fatal. A hope of recovery can only be conceived to exist in those cases (if there be, indeed, any such) in which the deposits are confined to one kidney, without implicating the excretory appendages. One does not see, *à priori*, why tuberculous masses in the kidney should not be evacuated by the urinary channels, in the same way that similar masses in the lungs are sometimes evacuated by the bronchial tubes, provided the tendency to the deposition of tubercle be arrested. Kidneys apparently undergoing a process of this sort have, in very rare instances, been found in the inspection of the bodies of persons who bear the marks of past tuberculosis. Dr. Bennett describes a case in which it appeared probable that such a train of events had taken place.[4]

If the disease involve both sides, or implicate the bladder and urethra, or be complicated with pulmonary or intestinal tuberculosis, no hope of a favorable issue can be entertained.

THE TREATMENT should be conducted on the principles which guide the management of tuberculous diseases generally. The strength should be supported by cod-liver oil, mineral acids, and other tonics, combined with a nutritious diet and a moderate allowance of stimulants. Opiates are generally required to insure rest and some alleviation of pain. These means may be supplemented by the occasional use of the warm bath. To check excessive secretion of pus, the muriated tincture of iron may be given in doses of 15 to 20 drops thrice a day (*see* Treatment of Chronic Pyelitis, p. 470).

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[1] Centralbl. f Med. Wissench., 1883, p. 65.

[2] Ibid., p. 145.

[3] To detect tubercle-bacilli, allow the urine to stand for a short time, and place a little of the pus between two cover glasses. Dry each glass carefully; stain like phthisical sputum, with aniline-water-magneta, decolorize with 25 per cent. solution of nitric acid. and stain the ground substance with methyl-blue. The tubercle-bacilli then appear red, while the other matters in the field of the microscope are blue.

[4] Clin. Lects., 2d ed., p. 734.

B.—SECONDARY TUBERCLE OF THE KIDNEY.

Secondary tubercle is deposited in the kidneys in the form of minute yellowish nodules and granulations, varying in size from a pin's head to a pea. The little masses are scattered over the surface and through the interior of the gland, chiefly in the cortical part. In places they run together into groups or patches as large as a sixpence or a shilling. The intermediate parts of the kidney are either altogether healthy, or only show signs of congestion immediately around the deposits. When the pyramids are affected, the little granulations sometimes evince a disposition to assume a linear arrangement parallel with the straight ducts. Such deposits are not uncommon in acute general tuberculosis; much less frequent in persons who have died from pulmonary or intestinal tubercle. The deposits are generally confined to the substance of the kidney, without participation of the pelvis and ureter.

Secondary tubercle is greatly more common in the kidneys than primary. Out of 91 cases of renal tubercle tabulated by Dr. Chambers, 76 were secondary, and 15 primary. *Both* kidneys are nearly always implicated in the former.

As a rule, no symptoms referrible to the kidneys are observed during life. The urine presents merely febrile characters, and contains neither pus nor blood. If, however, the deposit take place with excessive rapidity, pains in the back and other indications of renal disturbance may occur. In the following case, by Colin, deposition of tubercle in the kidneys, occurring in the course of chronic phthisis, was thus diagnosticated during life.

A soldier, aged twenty, suffering under chronic phthisis, was suddenly seized with violent lumbar pains accompanied with an intense rigor. Next day, these pains were so violent as to cause the patient to cry out; the lumbar muscles were in a state of strong contraction, and exquisitely tender. There was high fever, with a corresponding state of the urine. Three days later, acute meningitis set in, which destroyed the patient in four days. The autopsy revealed exudation of lymph (but no tubercle) on the meninges; old pulmonary mischief, with recent deposit of miliary granulations in the lungs; the spleen was studded with similar granulations. The kidneys were markedly enlarged, the capsule easily detached; about thirty yellow nodules, as large as pins' heads, were scattered on their surface. On the convex border of each kidney there existed, in perfect symmetry, two whitish patches about the size of a two-franc piece, composed of an aggregation of a large number of granulations identical with the preceding. Sections of the organs revealed an immense number of similar granulations scattered in the cortical substance, and to a less degree in the pyramidal portion. It was calculated that each kidney contained from 300 to 400 of these granulations. ("Gaz. Hebd.," x. p. 39.)

CHAPTER XIII.

ENTOZOA IN THE KIDNEYS.

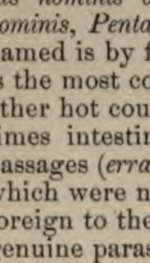
THE parasitic worms which infest the kidneys are: *Echinococcus hominis or hydatid*, *Bilharzia hæmatobia*, *Filaria sanguinis hominis*, *Pentastoma denticulatum*, and *Strongylus gigas*. The first named is by far the most common in these latitudes; the second is the most common in Egypt, Cape of Good Hope, and certain other hot countries; the last two are of extreme rarity. Sometimes intestinal worms wander into the kidneys and urinary passages (*erratic worms*); and in some notable instances, objects which were not parasites at all, or which were parasites wholly foreign to the human body, have been described and figured as genuine parasites of the urinary organs (*spurious worms*).

I.—HYDATIDS IN THE KIDNEY.

(*Echinococcus Hominis.*)

Hydatids in the kidneys are comparatively rare; they are much less common than hydatids in the liver and even in the lungs; they are more frequent than hydatids in the other organs and tissues of the body.[1]

FIG. 69.



Wall of a hydatid cyst, showing the laminated structure—not magnified. [After Davaine.]

NATURAL HISTORY.—A hydatid tumor consists of an adventitious *outer capsule*, composed of fibrous tissue, which is organically connected with the texture of the organ in which it is situated. Within this, and unconnected with it except by contact, lies the *hydatid cyst* itself. This latter varies in size from a walnut to an adult's head. The cyst-wall varies in thickness according to the size of the cyst, from about a line to a tenth of a line or less, and is composed of an opalescent tremulous substance resembling boiled white of egg. When examined more closely it is found to have a laminated structure (Fig. 69), and to be composed of an immense number of thin lamellæ or layers, which, under the microscope, exhibit a perfectly homogeneous

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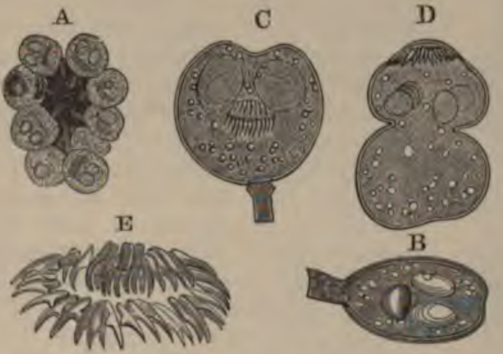
[1] Davaine gives the following rough approximations of the relative frequency of hydatids in the different organs and tissues:

Liver	166	Osseous system	17
Lungs	40	Parietes of the body	12
Kidneys	30	Heart	10
Pelvis	26	Orbit	9
Brain	20		

structure. Within the cavity of the cyst a number of secondary or daughter cysts float freely in a watery saline fluid, which is devoid of albumen.[1] The daughter cysts vary in size from an orange to a pea or pin's head: they may be even much smaller than this, and require a microscope for their detection. A mother cyst may, however, be barren: that is, contain only fluid contents; but this is rare. More commonly twenty, thirty, a hundred, or even many thousand secondary cysts float within it. The structure and attributes of the secondary cysts are identical, in every respect, with those of the parent; and their walls display the same characteristic lamination.

Sometimes this constitutes the entire anatomy of a hydatid cyst; but as a general rule additional structures are found, which indicate a more advanced phase of development: these are: a *germinal membrane* lining the interior of the cyst, and certain minute animalcules growing therefrom which are termed *echinococci* (*scolices* or *tænia-heads*).

FIG. 70.



Human echinococci. A. A group of echinococci, still adhering to the germinal membrane by their pedicles, magnified 40 times. B. An echinococcus magnified 107 times; the head is invaginated in the caudal vesicle; a pedicle is attached to it. C. The same compressed; the head retracted, the suckers and the hooks are seen in the interior. D. Echinococcus magnified 107 times; the head is protruded from the caudal vesicle. E. Crown of hooks magnified 350 times. [After Davaine.]

The *germinal membrane* is a thin, transparent, homogeneous, (unlaminated) tough membrane, which forms an interior sac closely applied to the inside of the hydatid vesicle. When detached and emptied it shows a tendency to contract and curl on itself in a peculiar manner.

The *echinococci* (Fig. 70) are minute ovoid animated beings, just visible to the naked eye. When magnified they are found to consist of a head resembling that of a tapeworm, provided with four suckers and a double crown of hooks (E). When the head is stretched out (D) it is seen to be connected by a short thick neck to a "caudal vesicle," which is somewhat larger than the head. The head is generally retracted within this caudal vesicle; and then the little body assumes a spheroidal figure with the crown of hooks in its interior (B C).

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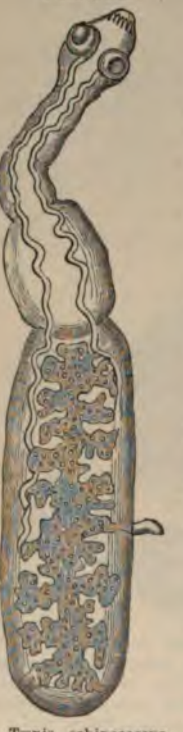
[1] This is not strictly correct. I have twice detected a not inconsiderable trace of albumen in the fluid of hydatid cysts of the liver.

The echinococci are developed on, or rather in, the germinal membrane. They grow in groups of six to ten individuals, and are at first encapsuled in the substance of the germinal membrane. As they increase in size they burst through their capsule, and are then found attached, each by a short stalk or pedicle, to the germinal membrane (A). By-and-by they break loose from this attachment and float at large in the hydatid vesicle, sometimes with a portion of their stalks still adherent.

FIG. 71.



Tænia echinococcus, magnified 22 times — [After Van Beneden.]

Both the echinococci and the germinal membrane are liable to perish (from inflammation or some other cause), and then only scattered hooks or shreds of membrane are found floating in the turbid contents of the hydatid vesicle.

A marvellous light has been thrown in recent years on the zoölogical position of these worms, chiefly by the researches of Siebold and Van Beneden. It has been ascertained that the hydatid worm found in man[1] constitutes the *encysted phase* in the development of a very minute tapeworm which infests the dog.

The tapeworm in question (Fig. 71) is the *Tænia echinococcus* of Siebold (*Tænia nana* of Van Beneden). The entire adult animal is so small that it scarcely exceeds the size of a millet-seed. It consists of but three segments, of which only the last is fruitful. When this segment arrives at maturity it is cast off and a new one developed in its place. Myriads of these worms are sometimes found in the intestines of the dog, and their eggs are discharged in countless numbers with the excrements. The eggs so discharged are scattered far and wide; and some of them find their way with the food into the stomachs of men and other creatures suitable for their further development. Arrived there, the embryo is liberated; and, after penetrating the mucous membrane, it burrows its way, or is carried by the blood current, to some distant organ, where it is arrested. Having thus lodged itself, it presently reappears as a hydatid vesicle, in which, finally, are developed the echinococci as before explained. Dogs in their turn become infested with the corresponding tænia by feeding on the offal of slaughtered sheep, pigs, etc., which had been infested with hydatids. The echinococci therein contained develop in their intestines into the tænia echinococcus: and so the circle of transformation and development recommences.[1]

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[1] The same species infests the pig, monkey, sheep, and ox.

In the records of medicine may be found some seventy or eighty instances in which hydatids existed in the kidney or were passed by the urethra. In a number of these, the fact is simply mentioned; but in sixty-three cases some fuller details are communicated, and from an analysis of these the following account is drawn up.

It is necessary to remark that when hydatids are discharged by the urethra, it may be assumed as almost certain that they are derived from a cyst situated in the kidney. In the great majority of the cases, proof of this was obtained either from the examination of the body after death, or from the plain indication of the symptoms during life. In some cases, however, this was not so; and it remained opened to conjecture whether the parent cyst was not situated in the vicinity of the ureter or bladder, and opened directly into those channels. Such an occurrence seems, however, extremely rare, and I have only been able to find one instance in which actual proof of this was obtained.[2]

Morbid Anatomy.—The left kidney is more frequently the seat of hydatids than the right: out of 42 cases, the left kidney was affected 22 times and the right 18 times, and both organs together only twice. The less liability of the right kidney depends probably, as Béraud suggests, on the larger bulk of the liver intercepting a greater proportion of the embryos which travel from the intestine rightwards, than the smaller bulk of the spleen does of those which travel leftwards. In rare instances, hydatids have been found in the liver and other organs as well as in the kidney.

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[1] For further information and details of experiments *see*—Gervais and Van Beneden, t. ii. p. 270 et seq.; Davaine, loc. cit., Synopsis, 7 and 24; and Siebold's memoir on tape and cystic worms, bound with the second vol. of Küchenmeister's Manual of Parasites. Syd. Soc.'s Translation.

[2] In the Med. Times and Gaz. for 1855, i. p. 161, a case is referred to, on the authority of Mr. Birkett, in which hydatids were withdrawn by catheter from the bladder. After death a large hydatid tumor was found between the bladder and rectum, pressing upon the neck of the former. Rayer (loc cit., iii. 354, foot-note) relates an instance in which a hydatid tumor in the left iliac fossa opened into the rectum, with expulsion of hydatid vesicles with the stools and discharge of pus and gas by the urethra. He cites another (p. 554, note), in which hydatids were passed by stool, and afterwards a large hydatid escaped by the urethra; but there is no information as to the seat of the cyst, the patients having recovered. There is another case, recorded by Mr. Fynney in an appendix to the second vol. of the Memoirs of the Medical Society of London, in which hydatids were passed with the urine from a cyst which in all probability existed between the bladder and rectum. Immediately before the discharge of the vesicles the patient felt something give way in the neighborhood of the bladder. The patient died in a few weeks; but the exact seat of the cyst was not verified by post-mortem inspection. Cases of this class can be distinguished from renal hydatids by manual examination through the rectum or vagina.

As a rule, the cyst is lodged in the substance of the kidney; sometimes, however, between the capsule and the gland. As the cyst grows it encroaches more and more on the renal tissue, and eventually may entail total destruction of the organ. It forms a roundish, elastic, fluctuating tumor, projecting from the surface of the kidney, and varying in size from an egg to an adult's head.

The cyst has a natural tendency to make its way toward the pelvis of the kidney, and discharge its contents by the ureter. When it is situated in the pyramidal portion, this event takes place early, before the cyst has attained any great dimensions: but, when situated in the cortical part, or beneath the capsule, the cyst may exist for years, and grow to a large size, before it bursts into the infundibula. It may even not burst at all; and, still more rarely, it may penetrate upward into the chest and be evacuated through the bronchi, or open into the intestines and be discharged by stool. Sometimes, after opening in one direction, it effects a second opening in another direction. In no instance on record has the cyst burst into the peritoneum. The following table exhibits the relative frequency of these various modes of opening in our 63 cases:

The cyst opened into the :[1]		
Pelvis of kidney	in 47 cases	Hydatids discharged by the urethra.
Pelvis of kidney and lungs . .	1 “	
Pelvis of kidney and intestines .	3 “	
Pelvis of kidney and stomach .	1 “	
Lungs alone	in 1 case	No hydatids discharged by the urethra.
Did not open at all	8 “	
Opened artificially	2 “	

Hydatid cysts of the kidney, like hydatid cysts elsewhere, are liable to certain accidents. They may contract adhesions to surrounding parts; occasion inflammation and abscess in their vicinity, and the cyst may burst into such an abscess. The cyst itself may suppurate; or it may perish, and its germinal membrane and echinococci be destroyed; the fluid it contains may then be absorbed, and the whole crumple up into a hard depressed nodule, which henceforth lies dormant and obsolete. This obsolescence may ensue without bursting of the sac, or it may follow complete evacuation of its contents. The contraction and obsolescence of a hydatid cyst are accompanied by deposition of a whitish cretaceous and sebaceous material between it and the adventitious capsule, and within its own cavity. This deposit was formerly erroneously supposed to be of a tuberculous nature. Under the microscope it is found to consist of amorphous phosphate of lime, crystals of triple phosphate, cholesterine plates, and fatty granules. Amid this débris, echinococci hooks and shreds of laminated membrane may be found.

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[1] No authenticated cases exist of a hydatid cyst of the kidney opening in the loins. Rayer (iii. 578) mentions two examples of hydatid cysts in the loins, which suppurated and burst externally in the lumbar region. He seems to infer that the cysts in these cases were connected with the kidney: both ended in recovery. It is more probable, however, that the cysts were lodged superficially in the muscular tissue of the lumbar region. In a later case of this kind which ended fatally, it was ascertained post-mortem that the cyst lay surperficial to the kidney and unconnected with it.

Hydatid cysts are also liable to external violence, especially when they form a palpable tumor in the flank. A blow or fall has in more than one instance been the apparent cause of the bursting of the sac into the pelvis of the kidney; and the patient has dated his symptoms from the occurrence of some such accident.

The opening of the cyst into the pelvis of the kidney is soon followed by the passage of secondary or daughter vesicles along the ureter into the bladder, from which they are expelled sooner or later with the urine.

The first of the two following cases illustrates the ordinary mode of evacuation by the ureter; the second by the ureter and lungs:

CASE 1. *Hydatid vesicles voided by the urethra, at intervals, for twenty years, with symptoms resembling nephritic colic. Hydatid cyst found in the left kidney* (Chopart, loc. cit., p. 78).—A young lady of 25 was seized with a violent pain in the left lumbar region, with all the symptoms of nephritic colic. There was difficulty of micturition, tension, and tenderness of the abdomen. The bladder was full of urine, but some obstruction prevented its flow, though there was constant desire to pass it. In the course of the night the emptying of the bladder was effected, with discharge of a large number of hydatids. When the discharged vesicles were examined on the following morning, the majority were found ruptured, and consisted of loose membranes only; some were entire, and contained a turbid fluid. The patient was relieved by the evacuation; but the pain returned again in less severity two days after. This pain commenced in the kidney, and when it diminished in that organ it increased at different points in the course of the ureter, and became more acute at the entrance of this canal into the bladder. When the hydatids had reached the bladder the pain in all these parts was replaced by a sort of lassitude.

The patient stated that she had been subject to similar attacks for twenty years; and that they always terminated in a discharge of little bladders full of water. Some of these were as big as a pigeon's egg; others were much smaller; the latter always came away first. The attacks recurred at irregular intervals; she was sometimes six months, a year, two, or even three years without an attack. In some of the attacks the efforts at micturition would be long unavailing, until at length, by increased effort and pressure on the belly, the hydatids would shoot out with a sort of noise, and then the urine followed in full stream. Four years later, the patient died; it is not stated from what cause. The left kidney was found converted into a thick and firm hydatid sac, filled with vesicles. The pelvis and ureter were greatly dilated. The right kidney was healthy.

CASE 2. *Hydatid cyst of the right kidney, which opened first into the ureter and subsequently into the right lung. Hydatid vesicles discharged with the urine and by coughing* (Béraud, loc. cit., p. 63).—Madame B., æt. 54, had experienced, for several months, pains in the right lumbar region and occasional difficulty of micturition; otherwise the health was good.

On August 30, 1851, she was suddenly seized with such violent pain in the right kidney that she was obliged to be carried home. M. Fiaux, who was called to the case, found extreme distention of the bladder; a catheter was introduced, and the urine withdrawn presented nothing unusual. The patient passed a good night, and was quite restored in a couple of days.

On September 15th, she went to St. Denis, where she was seized with the same symptoms as before. She succeeded, after great efforts, in expelling by the urethra a little membranous vesicle as big as a pigeon's egg, and immediately afterwards she passed abundance of water, and was relieved.

On the 26th, the pains returned; they commenced in the right lumbar region, and radiated towards the pelvis and the right thigh. She tried to pass water several times during the night, without success. The bladder reached almost to the umbilicus. A large quantity of clear urine was withdrawn by catheter with immediate relief.

From the 8th to the 23d of October, retention of urine recurred on three occasions, and the urine withdrawn did not present any peculiarities.

On November 2d, the pain in the kidney returned with great severity; it mounted to the liver and descended along the ureter to the thigh; there was thirst, hot skin, with tenderness and meteorism of the abdomen. The patient had passed water several times during the night, but in very small quantities. The urine was turbid, with a glairy deposit at the bottom of the vessel. From this date to the 22d, the pain diminished; the urine continued turbid, and contained pus.

On the 24th, the patient had a violent rigor, and the renal pain became more severe than ever. Vomiting occurred several times during the night, and three or four liquid stools were passed. She also voided urine several times. M. Fiaux now observed, for the first time, in the urine, shreds of membrane having the characters of hydatids. On examining the right flank, an oblong tumor was found below the liver, apparently united to it, extending to the iliac fossa and having a breadth of about

4½ inches. The tumor was hard, and tender on pressure; no loop of intestine passed in front of it; the lumbar region behind presented a tolerably prominent bulging. It was no longer doubtful that this was a hydatid tumor of the right kidney in a state of inflammation.

From this time hydatid fragments continued to be discharged with the urine from time to time, and the lumbar fulness became more pronounced.

On the 22d of December, under the advice of Gendrin, steps were taken to open the tumor, and several caustic issues were established on the front of it.

But on the 2d of January violent pain set in at the base of the right lung, with cough, mucous expectoration, and fever. Frequent shivering occurred the next day, and the pain and fever continued.

January 7.—The oppression was increased. Violent fits of coughing occurred, with abundant purulent expectoration, of a fetid urinous odor; and this was mingled with membranes similar to those discharged with the urine. She continued to cough up hydatid shreds and urinous pus and to become gradually weaker until January 22d, when she died in a fit of suffocation, after having discharged seven or eight hydatids.

Autopsy.—The small intestine was thrust to the left; the ascending colon bordered the tumor, and was intimately connected therewith in its lower two-thirds. The right lung was indurated at its base, and united to the diaphragm. Behind the cyst was a purulent collection, as large as an orange, which communicated with the cavity of the cyst. The liver, left lung, and stomach were healthy.

The tumor was found adherent to the lower surface of the liver. It was constituted by the right kidney, which was converted into a sac as large as a child's head. Few remains of the renal tissue were found. On cutting open the sac, it was found to communicate by two distinct openings with the dilated pelvis of the kidney and the abscess. The latter again, which occupied the vault of the diaphragm behind the liver, communicated by a perforation through the diaphragm with a ragged cavity in the base of the right lung. All these cavities, with the pelvis of the kidney, the bladder, and the bronchi, contained a purulent fluid and numerous hydatid vesicles.

In rare cases, the secondary cysts contain a tertiary series (granddaughter cysts). Baillie mentions such an instance in the body of a soldier, whose kidney was found to contain a large hydatid cyst. Some of the secondary cysts in this instance merely contained fluid; others contained small vesicles floating in their interior.[1]

Occasionally, crystals of uric acid have been found adhering to the expelled hydatids; and in Mr. Barker's case, to be presently related, Mr. Queckett found in the interior of some of the cysts crystals of triple phosphate, uric acid, and oxalate of lime. In four cases calculi were found with the hydatids in the kidney or bladder; or were passed by the urethra.

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[1] Baillie, Morbid Anat., 5th ed., p. 294.

The Symptoms differ essentially according as the cyst has forced a passage for its contents into the pelvis of the kidney, or elsewhere, or still maintains its integrity. In the latter case the cyst remains wholly latent until it attains sufficient bulk to form a palpable tumor in the flank. As the tumor grows, it displaces the viscera in its neighborhood, generally without further mischief; but sometimes inflammatory adhesions or suppuration take place in its vicinity and occasion intercurrent attacks of pain and feverishness. In eighteen out of our sixty-three cases, tumor in the side was discernible during life. It varied in size from an orange to an adult's head, and presented a rounded form and an elastic feel. In some instances fluctuation was distinctly perceived in it; in others obscurely; in others not at all. The peculiar thrill characteristic of hydatid tumors (hydatid fremitus) was observed only in a few instances. In order to evoke this sign, the fingers of the left hand should be laid upon the tumor, and tapped sharply with the fingers of the right. A thrill is then communicated to the overlaid fingers, which has been compared to the vibrations of a repeater watch held in the hand. A similar sensation is communicated to the ear when the stethoscope is applied and the tumor tapped with the fingers.[1] Sometimes the fremitus is absent under conditions which appear favorable to its production. In a case reported by Livois (cited by Béraud) even Rayer was unable to detect anything beyond ordinary fluctuation, and diagnosed a hydronephrosis. After death the kidney was found converted into an enormous hydatid sac containing multitudes of secondary vesicles, varying from the size of a grain of millet to a hen's egg.

The topographical characters of the tumor agree with those of renal tumors in general. The colon is usually found in front of the intumescence: but it is important to know that this is not invariable. Béraud communicates a case from Nélaton's clinique, in which the descending colon ran along the outside of a hydatid tumor of the left kidney: in Fiaux's case, already related (p. 564), the ascending colon coursed along the inner border of the tumor, and no intestine separated it from the abdominal parietes.

When the cyst bursts into the pelvis of the kidney, the escape of its contents by the urethra constitutes a capital symptom. This may occur with or without symptoms referrible to the renal region (tumor, nephritic colic, etc.). Entire vesicles mixed with broken ones are usually voided; in other cases only fragments are passed, or a milky detritus in which echinococci hooks, laminated shreds, and oil particles may be detected by the microscope.

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[1] The history and theory of the hydatid fremitus may be found discussed at length (with an account of Davaine's experiments) in Meissner's Beiträge zur Lehre von dem Vorkommen des Echinococcus, etc. Schmidt's Jahrb., Bd. 116, S. 183.

The discharge of vesicles takes place in paroxysmal attacks at wholly irregular intervals. In exceptional cases, only one paroxysm is experienced, during which the cyst is seemingly entirely evacuated, and then finally contracts. In the great majority of cases, however, the first attack is succeeded by many others. The interval between them may be a few weeks, or a few months, or many years. In a case reported by Tomowitz, the second attack occurred three years after the first. In Quinquerez's case, seven years elapsed between the first and second discharge of hydatids; then the attacks followed each other more frequently, at intervals of one or more years, for ten years; in the last year they recurred every four or six weeks.

An attack is usually ushered in by sharp pain in the loin, sometimes with a sensation as of something giving way internally. The pain shoots down along the ureter to the inside of the thigh. It may be attended with rigors, sickness, and hiccough—though this is rare; then follow colicky spasm in the course of the ureter, indicating the descent of vesicles along that canal—sometimes aggravated by suppression of urine and retraction of the testicle. These symptoms continue a few hours or several days, and they commonly cease suddenly, often with a feeling as if something had dropped into the bladder, The urethra is next forced, and new symptoms arise—retention of urine, excessively frequent desire to pass water, with severe pain extending to the end of the penis. When the vesicles are expelled, relief follows. The number of vesicles discharged during an attack varies from one or two to several dozens. The urine is often tinged with blood or mixed with pus. The force required to effect the final expulsion is sometimes sufficient to propel the vesicle a considerable distance with an audible thud.

The paroxysms are sometimes determined by some evident exciting cause, such as a blow or fall, or by horse or carriage exercise. In Zinkeisen's case the attacks usually followed the use of spirits and strong coffee.[1]

After each discharge of hydatid vesicles the tumor (if any exist) may subside sensibly. On the other hand, rapid enlargement of the tumor, from distention of the pelvis with accumulated urine, may follow the impaction of a vesicle in the ureter. Repeated discharges occasion dilatation of the passages, and enable the patient to void larger vesicles with less pain.

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[1] Schmidt's Jahrb., Bd. 116, S. 290.

The following examples illustrate the eccentric course and usual symptoms of renal hydatids:

CASE 3. *Repeated discharge of hydatid vesicles with the urine; tumor in the left lumbar region—final recovery* (Lettsom, "Memoirs of the Medical Societies of London," vol. ii. p. 32).—A gentleman, aged 32, was thrown off his horse in February, 1780, and received an injury in the lumbar region. This was followed by considerable hæmaturia. In a fortnight all the consequences of his fall had disappeared; but in the following June he spat blood; this also passed rapidly away. Three years later, he was seized with shivering and a violent pain in the left lumbar region. A few days after, he perceived an enlargement in the hypochondrium. This increased gradually until February, 1784. After the first month the tumor was so little painful that he was enabled to take a journey of 130 miles to London to consult Dr. Lettsom.

A fluctuating tumor as large as an infant's head was detected in the left hypochondrium, extending from the spine to the umbilicus and from the ribs to the os innominatum.

As the swelling augmented the pain increased, and the patient suffered considerably from the action of walking and from motion in general. At length (February 20th) some difficulty in making water was experienced, and for many hours there was a total obstruction of urine. The same night there was great pain with violent rigors; but early in the morning the patient experienced the most happy relief by the discharge of a large quantity of thick pus with the urine, which was followed the next day by the escape of numerous hydatids.

In a few days the tumor subsided, and the purulent discharge ceased; after this, he continued recruiting his health for nearly a fortnight, when his side enlarged again, after exercise in a coach, probably by a large hydatid stopping up the ureter; rigors and strangury succeeded as before, and the tumor became as large as in the first instance, until the latter end of March, when he experienced a second discharge in every respect like the former, excepting that the hydatids were larger.

His health and strength again returned, until his side filled a third time, after exercise on horseback, and continued swelling until the 25th of April, when he was again relieved by a third discharge; the hydatids now passed were considerably larger than those of the preceding attacks.

The passages now became so open, that he frequently discharged hydatids after walking or riding, without enlargement or pain of the side; or if he felt uneasy or perceived a tendency to tumescence, by pressing his hand upon his side he could squeeze the vesicles into the bladder, where they would remain some time before they were discharged; but the hydatids became at length so considerable in size that it was with great difficulty they passed the urethra. The last vesicle which he voided (on the 12th of July) was so very large that it stopped up the urethra, and remained in it for a considerable time, until the weight of the accumulated urine forced it away.

The earliest hydatids voided burst in their exit; and they gradually increased in magnitude in every successive discharge; the first which he passed were not bigger than the skin of a green pea, and the last about the size of a pullet's egg.

Since this last discharge his health was gradually reëstablished; he was able to enjoy, without the least inconvenience, thereafter, the chase and every other species of exercise as well as ever he did.

CASE 4. *Discharge of hydatids by the urethra in periodical paroxysms occurring yearly for a period of thirty-seven years* (Vigla, "Bulletin de la Société Anatomique," 1838. Cited by Béraud, loc. cit., p. 57).—A healthy woman, æt. 37, had suffered from her infancy with her present symptoms, which occur in annual paroxysms. Every winter, and generally in the month of January, she experiences in the left renal region a pain, which speedily becomes severe and forces her to relinquish her occupation. There is no fever; nor vomiting; but the appetite is lost; the urine remains natural. At the end of two or three days of this condition, she voids a very large number of hydatids, mingled with a turbid urine. This emission takes place two or three times a day, for three or four days, and then she returns to her ordinary health. Sometimes, but very rarely, similar attacks occur in the course of the year, but slighter; these latter consist of a violent pain in the same place, not lasting more than two or three hours, at the end of which an emission of urine with discharge of hydatids ensues, and the pains disappear. These slighter attacks have never recurred more than once or twice in the same year. In the year 1828 one of the annual January attacks was observed by M. Vigla. The prodromata—that is to say, the pain and uneasiness—were accompanied with feverishness; but this was attributed to a coexisting acute pulmonary catarrh. At the end of four days, as usual, the emission of urine charged with hydatids commenced, and continued for four days. The quantity of vesicles which she rendered was enormous; for she passed urine twice or thrice on each of these three days, and every time from 40 to 50 large hydatids were found in the urine, without counting the little ones. The larger ones passed the first, but ruptured and empty; the largest of all surpassed the size of a pigeon's egg. The smaller ones were voided entire, and full of a semitransparent fluid; some were smaller than a pea. There were no symptoms referrible to the bladder.

CASE 5. *Hydatid cyst of the right kidney; suspicion of pregnancy; discharge of hydatid vesicles by the urethra* (Babington, "Med. Times and Gaz.," 1855, i. p. 160).—A healthy single woman, æt. 27, was admitted into Guy's Hospital on February 8, 1854. About the age of 23 she was one day kicked in the abdomen by a child which she was carrying upstairs. The kick gave her much pain, and on the night following she discovered for the first time a tumor about the size of an egg in her right side. The tumor gradually increased, and in the course of a year became so large as visibly to distend the abdomen. About this time the menstrual function was suspended, and the increasing size of the abdomen caused her great trouble, by exciting suspicions in the minds of her relatives that she was pregnant. The enlargement, however, continued beyond the usual period of utero-gestation, and anxieties as to the nature of the disease took the place of the suspicions alluded to. She was now sent up to London from her home in Oxfordshire, and was admitted into St. Bartholomew's Hospital under Dr. Hue. The tumor was at this time stationary, and her general health good. After a few weeks' stay in the hospital she was discharged, and returned to service, where she continued without material change in her condition until about a year prior to her admission into Guy's Hospital, under Dr. Babington, when she began to pass "skins and little bladders," with the urine. These bodies continued to be voided afterwards in large numbers. Often a vesicle would get impacted in the urethra, and require to be pulled out with the fingers. At first neither blood nor matter was ever present in the urine. About two weeks before admission, however, after having been confined to bed for several days with intense pain in the side, she suddenly felt a sensation as if something burst within her, and shortly afterwards matter and blood began to escape by the urethra. The tumor had meanwhile much diminished in size, and at the time of her admission there was no visible enlargement of the abdomen. During her illness she had lost some flesh, but still retained a fairly robust appearance.

On examination of the abdomen, a large mass apparently about the size of a fœtal head, but flattened, was easily felt in the right hypochondriac or lumbar region. It was not tender, and felt firm. The patient remained under Dr. Babington's care for several months, during which vast numbers of hydatid vesicles were passed. The vesicles varied much in size, some were broken and others whole. The urine contained also much pus. The girl somewhat improved in health, and the tumor became decidedly smaller before she left the hospital; at the time of her discharge, she still continued to void occasionally pus and hydatid vesicles.

CASE 6. *Frequent discharge of hydatid vesicles by the urethra—nephritic colic and suppression of urine. No tumor in the flank* (Dr. Barker, loc. cit., p. 5).—A young man, æt. 28, came under the notice of Dr. Barker, of Bedford, on December 17, 1853. He was suffering from a dull aching pain in the loins, particularly on the left side, with frequent desire to pass urine, and slight difficulty in voiding it. The urine was healthy. On the 22d of December he experienced greater difficulty than ever in passing urine in the early part of the night, and for some hours he was unable to pass a single drop. Early in the morning he passed four little hydatid cysts with immediate relief. Subsequently he recovered sufficiently to follow his occupation during the summer of 1854, suffering nothing more than an occasional frequent desire to void urine.

On September 10, 1854, he passed six cysts; but with less pain than on the previous occasion—a result which the patient attributed to taking 10 drops of oil of turpentine, which had been recommended to him, and which greatly increased the diuresis. The urine after the passage of the cysts was tinged with blood.

On November 16th he passed four cysts. The passage of these was preceded by severe pain in the left kidney, by the passage of several pieces of clotted blood, and by considerable difficulty in voiding urine. Indeed, for two entire days he passed no urine. On this occasion he took 19 drops of turpentine, within two hours, in divided doses. Shortly after taking the turpentine, the pain in the left kidney suddenly ceased, with a sensation which, to use the patient's own words, seemed to indicate that "something had suddenly broken in the kidney." He then complained of pain along the left iliac region, which continued for several hours, and ceased as suddenly as the previous pain had done. After this, all attempts to void urine were accompanied with pain along the urethra, premonitory to the expulsion of the cysts from that passage.

He continued in good health, with the exception of occasional dull aching pain in the lumbar region, especially the left side, until December 9th. He then passed five cysts, but all smaller than the previous ones; and no more were passed until December 31st, when he awoke in the morning with acute pain in the loins, and all the symptoms previously described as occurring on November 16th. During the day he passed twenty cysts—one at 8 A. M.; eleven at 1 P. M.; five at 7 P. M.; and three at 11 P. M. The cysts passed in rapid succession, and some were of a size as large as a small walnut. On January 1, 1855, a single cyst was passed in the morning; on the 2d two others; on the 3d one; and on the 10th two. From this last date up to December 8th (beyond which the history is not carried), he continued to suffer frequently from attacks of pain and difficulty in passing urine, followed often by the expulsion of cysts, between seventy and eighty of which he brought to Dr. Barker.

Careful examination failed to detect any abdominal enlargement.

The urine often contained a small quantity of blood during and after the expulsion of the cysts; it was often loaded with lithates and phosphates; occasionally, crystals of uric acid were found attached to the outer surface of the cysts. The general health suffered little. Altogether upwards of 150 cysts were passed; they varied in size from a pin's head to a walnut. The larger vesicles contained echinococci; but many of the smaller ones did not contain any.

The *duration* of the symptoms is altogether uncertain. In some of the cases permanent recovery followed one or a few discharges of vesicles. Other patients went on passing hydatids for three, ten, twenty, and even thirty years. A discharge of vesicles having once taken place, there are no means of ascertaining whether any more will follow. Neither the number of vesicles voided, nor the frequency of the discharges, supplies any reliable indication. The only sign of value is the lapse of time since the preceding attack: the longer the interval the less probability of recurrence.

The usual *termination* is recovery. Out of sixty-three cases, recovery was assumed to have taken place in twenty; in most of these the attacks had ceased for some years. In sixteen cases, vesicles continued to be discharged at the date of the record; in nineteen cases the termination was fatal; and in eight we are left without information. Of the nineteen fatal cases, death took place in nine from causes other than the hydatid disease (phthisis, cancer, gangræna senilis, etc.); so that only in ten (sixteen per cent.) was the fatal issue attributable to the parasite. Death was brought about in these ten cases in diverse ways—by bursting of the cyst into the bronchi, by pleurisy from pressure of the tumor on the thoracic cavity, suppuration of the sac, etc. In a case reported by Dr. Blackburn, the left kidney was the seat of a hydatid cyst which had burst into the pelvis of the organ, where a large calculus was also found; the right kidney was congenitally absent: so that the abrogation of the function of the left (and unique) kidney proved necessarily fatal.[1]

Etiology.—Hydatids are not uncommon in England, France, and Germany: more rare in America and India. There is, however, no country so fearfully infested therewith as Iceland. According to Eschricht (speaking of hydatids in any part of the body), a sixth part of the population are afflicted with this parasite. The frequency of the disease is due to the vast number of dogs in that country, which live in intimate contact with the inhabitants, and are greatly infested with the tænia echinococcus. The ova of the parasite, discharged with the excrements of the dogs, foul the dried fish which forms a large part of the food of the population. The embryo of the parasite thus finds its way into the stomach, and thence travels into different parts of the body, giving rise to hydatid cysts.

The use of uncooked meat and salad is evidently an easy source of infection, in places where dogs are numerous and live in close intercourse with their masters. Dr. Barker's patient had been for a year a vegetarian.

Men appear more subject to renal hydatids than women—the proportion, in our sixty-three cases, was forty-one men to twenty-two women. In only one instance was more than one member of a family affected: in that case, a husband and wife passed hydatids by the urethra.[2]

The mean age, in forty-seven cases, was thirty-four years: the youngest was only four years, and the oldest seventy-five.

The Diagnosis presents no difficulty when a tumor exists in the side and hydatid vesicles are voided with the urine. When the vesicles are broken in the passage, the laminated structure of the pieces, or the finding of echinococci-hooks, decides the nature of the discharge.

So long as the parent cyst remains intact, the urine preserves its normal characters, and the diagnosis turns on the characters of the tumor in the flank. Hydatid fremitus, when present (which is rare), is a valuable sign; but its absence, as we have seen, has little significance.

Hydatid tumor of the kidney is most liable to be confounded with hydronephrosis; and in the absence of discharge of vesicles, or their débris, with the urine, and of hydatid fremitus, the diagnosis is extremely difficult or impossible: it rests chiefly on the indications of the previous history.

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[1] Lond. Med. Journ., 1781, p. 126.

[2] Gay, Med. Times and Gaz., 1855, I. 160.

When vesicles are voided with the urine, and no tumor can be detected in the flank, the seat of the parent cyst is sometimes indicated quite clearly, to be the kidney, by signs of nephritic colic—in other cases, more obscurely, by pains in the back and loins or about the crest of the ilium. When these indications fail, a careful examination of the pelvis should be made through the rectum or vagina: if no evidence be found of a tumor between these parts and the bladder, it may be inferred, almost with certainty, that the parent cyst is situated in the kidney.[1]

THE PROGNOSIS is generally favorable—much more so than in hydatid cysts of other internal organs (the uterus excepted), on account of the facility and safety of evacuation by the urinary passages. It is most favorable of all when the discharge of hydatids by the urethra is unassociated with tumor in the abdomen. In no such case has a fatal result been recorded: the cyst in such cases may be inferred to be small, and to be situated in the pyramidal structure of the kidney, whence its contents find easy exit through the infundibula.

When a renal tumor exists, the issue is still likely to prove favorable if the cyst has opened into the urinary passages. There is, however, some risk that a second opening may be formed in a less safe direction (into the lungs), or that the cyst, or the parts around, may suppurate. This latter contingency is by no means rare, nor is it necessarily fatal. In several instances large quantities of pus were discharged with the vesicles, and yet the issue was favorable. In three cases, in which vesicles were discharged both by stool and with the urine, the termination was favorable. In an instance recorded by Fleckles, a woman who had had a tumor in the side for many years voided frequently hydatids by the urethra, and subsequently a large quantity by vomiting. At the date of the report the case was going on favorably.[2] In the two cases in which the cyst burst into the cavity of the thorax, the termination was fatal.

When the cyst fails to open a passage for its contents into the pelvis of the kidney, the prospects of the patient are much more serious. The tumor is liable to attain very great dimensions, and, by its pressure, to excite inflammation in the surrounding parts, or within the chest; or the cyst itself may suppurate and be transformed into a vast abscess. The operation of puncturing such a cyst is one of considerable danger.

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[1] Prof. Otto Spiegelberg (Arch. für Gynäkol., i. 1, p. 146, quoted in Schmidt's Jahrb., Bd. 146, 1870) records the case of a woman, æt. 42, who for fifteen months had had a tumor in the right hypogastrium. It was movable, distinctly fluctuating, and about the size of a man's head; and extended downwards towards the brim of the pelvis. It could be felt and moved from the vagina, and was taken for an ovarian cyst. On proceeding to perform ovariotomy, when the cyst was opened, two echinococcus membranes escaped, which cleared up the diagnosis. A portion of the kidney was left behind. The patient died 26 hours after the operation.

[2] Schmidt's Jahrb., Bd. 87, S. 205.

TREATMENT.—The indications to be held in view are, to destroy the life of the parasite, to facilitate the evacuation of the cyst, and to combat the accessory symptoms and complications.

Whether medicines administered internally have any real power to destroy the life of a hydatid parasite, or to facilitate the evacuation of a hydatid cyst, may be greatly doubted. Nevertheless, oil of turpentine has obtained a certain reputation on the strength of its tæniafuge properties. The echinococcus of the hydatid vesicle is undoubtedly identical with the head of a certain species of tapeworm; but the condition of a parasite free in the intestinal canal, is widely different from the encysted state of the same parasite in the substance of the kidney, where remedies can only reach it indirectly, by the circuitous route of the circulation. Turpentine was given in a large proportion of the recorded cases; but there is little evidence that it had any beneficial influence beyond its diuretic effects.

The escape of the vesicles in Dr. Babington's case, was thought to be favored by a course of iodide of potassium.[1]

A variety of other vermifuge and diuretic medicines have been used, with more or less show of success—calomel, nitrate of potash, the caustic alkalies, hemlock, taraxacum, etc. Béraud states of his patient, that whenever he took white wine, and beverages containing nitre, he voided a much larger number of vesicles than at any other times. On two different occasions he was made to take 20 grains of nitre in dandeloin tea, and each time the desired effect was speedily produced.[2]

Electro-puncture has also been practised with a view to kill the worm; but without evidence of success.

When the cyst has opened into the pelvis of the kidney, the practitioner is able, in diverse way, to facilitate the expulsion of the vesicles, and to moderate the severity of the accompanying symptoms. Anodynes, especially opium, the warm bath, free use of diluents, are indicated during the passage of the vesicles; if the nephritic paroxysm be intense, blood may be abstracted from the loins by cupping. Sometimes mechanical aid is required to assist the liberation of the vesicles. Dr. Lettsom's patient helped their transit along the ureter by pressing them forward with his fingers; and in several cases it is noted, that patients (mostly women) have used the fingers to dislodge vesicles impacted in the orifice of the urethra. The use of the catheter is sometimes required to relieve the retention of urine caused by vesicles engaged in the urethra or pressing against the neck of the bladder.

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[1] In two cases of hydatids of the liver, I have seen the cyst gradually and completely contract after a course of very large doses (30 grains t. d.) of iodide of potassium.

[2] Gaz. d. Hôp., Aug. 11, 1832. Béraud, loc. cit., p. 93.

When the cyst remains closed, measures should be taken to evacuate its contents. In similar cysts of the liver I have, in four cases, adopted the following plan with uniform success. I employ a tubular gold needle—like the needle employed in subcutaneous injections, except that it is about twice as long. The base of the needle is mounted on a piece of India-rubber tubing three feet in length, and furnished with a small stopcock at its lower end. The tube is first filled with water by suction, and the stopcock closed. The needle is then thrust into the most prominent part of the cyst, and the lower end of the tube is placed in a vessel on the floor. When the stopcock is opened, the fluid begins to run—the column of liquid in the tube acting after the manner of a siphon, and exercising a soliciting force on the contents of the sac.[1] The wound made by these fine needles is so minute that there is no risk of extravasation into the peritoneum—and certainly no risk of peritonitis, as I have tested in a large number of instances. In two of my cases simple puncture by the needle has been sufficient, with only a withdrawal of a drachm or two of the contents, to destroy the life of a hydatid parasite, and to cause it to pass very gradually into obsolescence and absorption.[2]

It must, however, be remembered that evacuation of a renal hydatid is not so urgently called for as the evacuation of a hydatid of the liver; because in the former there is a natural tendency to spontaneous evacuation by the urinary channels, with very little risk to life, whereas, a hydatid of the liver has no such ready means of escape.

II.—BILHARZIA HÆMATOBIA.—*Cobbold.*

(*Distoma Hæmatobium—Bilharz.*)

This parasite was discovered by Bilharz, while conducting, with Griesinger, an investigation into the diseases of the Egyptians. Bilharz named it Distoma Hæmatobium; but later writers have erected it into a separate genus, which Cobbold has named Bilharzia in honor of the discoverer. It is an elongated, soft-skinned, bisexual entozoon, three or four lines in length, of the trematode or fluke kind (Fig. 72). It inhabits the branches of the portal system, and the minute veins of the pelvis of the kidney, ureter, and bladder. So common is it among the Egyptians, that Griesinger found it 117 times in 363 autopsies.

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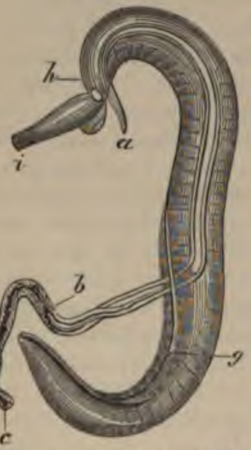
[1] This plan is substantially like an operation with the more recently introduced aspirator, but the force used is of more gentle and safer character,—*see* a paper by the author "On Exploration and Tapping" in the Liverpool and Manchester Medical and Surgical Reports for 1873.

[2] For a case of hydatids of the kidney successfully treated by aspiration, *see* Brit. Med. Journ., 1877, ii. p. 471.

FIG. 72.



Bilharzia Hæmatobia, highly magnified. *h i g*, the male; *a b c*, the female. [After Bilharz.]

The male (*h i g*) is comparatively thick and short, and provided with a gynæcophoric canal, in which the longer, filiform female (*a b c*) is lodged during the copulatory act.

The egg (Figs. 73 and 74) are oval bodies, $\frac{1}{170}$ of an inch long, with a spiny projection from the anterior end. The embryo, when newly escaped, is flask-shaped, and provided with cilia (Fig. 74).

This creature does not produce much mischief in the larger veins; but when lodged in the smaller vessels of the mucous and submucous tissue of the urinary and intestinal tracts, it engenders severe and often fatal disorganization. Griesinger found that, in the large intestines, it gave rise to a disease resembling dysentery, and that it was a frequent complication of that disease, but not the essential cause of it.

The ravages of the Bilharzia produce much more serious results in the urinary channels than in the intestines. It chiefly affects the bladder, but frequently also the ureter and pelvis of the kidney.

In the bladder, it gives rise to injected and ecchymotic raised patches, varying from the size of a lentil to that of a shilling, covered with a tough mucus, or with grayish-yellow, bloody exudation, which contains masses of ova. In more advanced stages the patches are more elevated, discolored, mixed with pigment specks, smooth and leathery, or soft, friable, and encrusted with gravelly matter, composed of uric acid and other urinary deposits, mixed with ova and blood. In other cases, the patches resemble nodules or condylomata, over which the mucous membrane is sometimes preserved uninjured, sometimes thickened, injected, adherent, or detached.

When the parasite invades the ureter and pelvis of the kidney, its effects are still more destructive. The calibre of the ureter is narrowed at the affected spot. Above the constriction, the ureter is dilated from accumulation of urine; the pelvis is also distended, and a hydronephrotic condition is produced. Or, inflammation and suppuration are set up, and severe pyelitis ensues. In one instance, Griesinger found the kidney distended into an enormous sac filled with pus—the renal tissue being wholly destroyed.

In addition to these direct results, urinary concretions are often formed on masses of ova, and grow into large calculi. This accounts for the frequency and endemic prevalence of calculous disorders in Egypt.

Griesinger remarks: "These various changes in the mechanical state and nutrition of the uro-poietic apparatus fail not to react most deleteriously on the entire organism. A series of cases have fallen under our notice, in which they produced general ill-health, and, at length, death. Most of these individuals were finally cut off, with shattered constitutions, by pneumonia, dysentery, and the like. . . . The direct signs of the disease are to be sought in the uro-poietic system, but especially in the urine. Repeated hæmaturia in sickly individuals, from unknown causes, often came before us in Egypt. We no longer doubt that the symptoms were produced by distoma-processes. The eggs of the distoma were found by Bilharz in the urine of a boy who, during convalescence from typhus, suffered from hæmaturia.[1] Symptoms of pyelitis, or slight affection of the bladder, must be present in many cases." . . . "Cases also came before us which awoke a strong suspicion that the Distoma disease sometimes runs its course as an acute, severe, and painful disorder. We found on two occasions, in the bodies of persons who had rapidly died from an unknown acute disease, abundant recent distoma-changes in the bladder, recent pyelitis, and a uniform dark-red hyperæmia of the kidneys. In other cases of supposed rapid typhus, the same changes were found in the bladder and ureter." These researches open a wide field for conjecture. Not only may urinary derangements and uræmia be occasioned by the ravages of the parasite, but septic infection may arise from the accumulation of heaps of dead and dying animals in the portal vessels; or the animals may creep into the general circulation, and find their way into organs of vital importance; in one instance distoma eggshells were found in the blood of the left ventricle.

In a note to his remarkable paper, Griesinger throws out the conjecture that the endemic hæmaturia of hot countries may be due to the presence of this worm in the urinary passages. A most interesting confirmation of this conjecture has been supplied by the researches of Dr. John Harley. Dr. Harley had an opportunity of examining the urine of three gentlemen who had resided at the Cape of Good Hope, and who had been subject to the endemic hæmaturia of that country. One of them still continued to be affected with slight hæmaturia; the other two considered themselves cured of the hæmaturia, but were subject to gravel. In the deposit from the urine of all three, Dr. Harley detected numerous ova of the Bilharzia. The condition of the urine in the first case is thus described by Dr. Harley: "Pale-amber colored, sp. gr. 1017.6, acid, deposits a deep layer of dirtyish-white flocculent matter, amongst which were two short opaque filaments about the $\frac{1}{30}$ of an inch in diameter, of a brownish color and soft consistence, two shorter and wider fragments of the same substance, a little reddish mass of the size of a hemp-seed, like a little clot of blood, and numerous white specks. The clear limpid urine, when acidulated with nitric acid and heated, deposited a trace of albumen." Uric acid, oxalate of lime, and urates were also sometimes found. The deposit, examined microscopically, was found to contain pus corpuscles; and the filamentous bodies and coagula contained embedded in them great numbers—sometimes thirty or forty, or more—of bright, highly refractive, oval bodies, which were identified as the ova of Bilharzia (Fig. 73). These observations seem to establish the parasitic origin of the endemic hæmaturia of Cape Colony, and render it extremely probable that the endemic hæmaturia of Mauritius and other hot climates has a similar origin.

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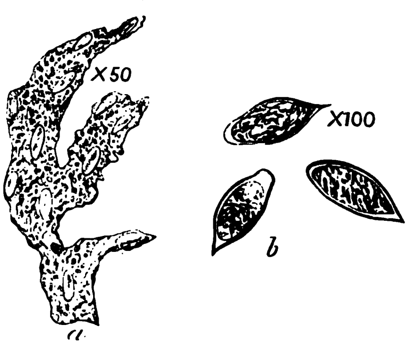
[1] Griesinger states that the clinical aspect of the subject only began to engage their attention when he and Bilharz were about to quit Egypt.

FIG. 73.

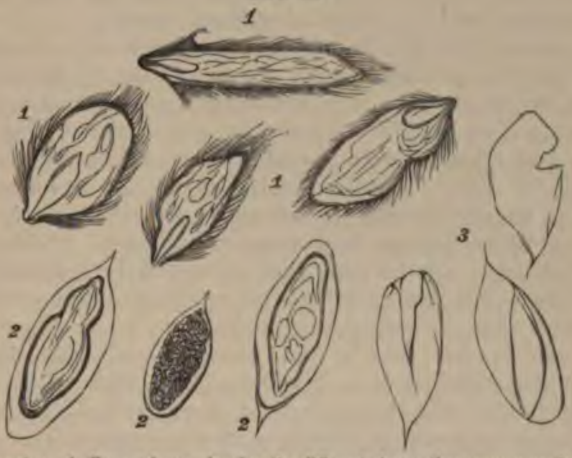


Ova of Bilharzia hæmatobia, found in the urine of a patient suffering from the endemic hæmaturia of the Cape of Good Hope. *a*, filament of mucus containing ova embedded, × 50; *b*, ova as they appeared in the fresh urine, × 100. [After Harley.]

An example of this curious disease came under my notice in the person of a groom, who had been in the employ of the Viceroy of Egypt. I am indebted to my friend Dr. Simpson for an opportunity of examining the case.

William Ray, æt. 19, was admitted into the Manchester Infirmary under Dr. Simpson, in February, 1871. He stated that rather more than two years ago he went to Cairo, as groom in the service of the Viceroy of Egypt. After a stay of some months, he went to Alexandria for the summer, returning to Cairo in the winter. He returned to this country about four months ago. While in Egypt he had been in the habit of drinking the water of the Nile unfiltered, and of eating water-cresses freely; with one or two exceptions, he invariably rode his horse bare-backed. About four months after his arrival in Cairo, he observed that he passed bloody-looking urine, and shortly afterwards he suffered pain in the back and perineum when riding. Since then he has persistently passed turbid urine containing blood. He is now very anæmic and thin, but in fair health apart from the urinary affection. On the 10th of March I examined the patient's urine. The specimen was a fair sample of what is generally passed. It was smoky and turbid, with an abundant reddish-white deposit in which might be seen little flakes of blood-clots; it was neutral to test-paper, specific gravity 1010, and contained a little albumen—rather more than the blood and pus would account for. Under the microscope, the deposit was seen to consist mainly of pus, mixed, however, with blood, both in the form of shreddy clots and as free corpuscles. Both ova and free embryos of the Bilharzia hæmatobia were present in considerable numbers (*see* Fig. 74). The embryos, in the mature ova (2), exhibited slow expanding and contracting as well as oscillating movements; these expansile movements were especially seen in the cervical narrowing, which at one time became very marked, and anon was almost effaced. The free embryos (1) moved actively in the urine for some hours after its emission. They were covered all over, with the exception of the head, with

FIG. 74.



Bilharzia in urine. 1. Free embryos, showing the different shapes they assume as they swim about in the urine. 2. Ova containing unhatched embryos. 3. Empty shells from which the embryos have escaped.

long vibrating cilia, by means of which they moved in various directions, with intervals of quiescence. At times an embryo could be seen racing across the field of the microscope with such speed that the eye could scarcely follow it—at other times, the movement consisted in stretching out the body to its full length, and then retracting it to an oval or ball-shape. When moving across the field, the body turned over and over on its longitudinal axis. Sometimes the head was retracted into the interior of the body. A number of empty eggshells, with irregular openings in them, or in fragments, were also seen scattered over the field.

This patient continued under observation for two or three weeks, and then left the hospital without leave.

In this case all the ova had terminal spines—not lateral ones—so that they resembled exactly the ova found by Harley in the cases from South Africa. Dr. Harley states that in his cases he never "met with a free living embryo in the urine. Eggs, which split open and liberate active embryos immediately after they are placed in water, remain quiescent for an indefinite time when left in the urine, and all attempts to hatch them in this fluid kept fresh and warm had invariably failed" ("Med.-Chir. Trans.," vol. 54). In the case just recorded this was certainly not the case—the embryos moved actively in the urine for several hours after its emission.

It does not appear that a man affected with this disease is capable of transmitting it to his wife. The wife of one of Dr. Harley's patients had three or four healthy children, and the husband had been passing numbers of the eggs of the parasite every day of their married life—yet the lady had never had the slightest symptoms of the parasitic disease, and the urine was free from all traces of the parasite. It seems probable that the parasite usually gains entrance into the body through the stomach by means of drinking infected water or eating salad, to which the minute animal or its embryos or ova adhere. Dr. Harley suggests that it sometimes obtains admission through the skin, that the minute leech-like animal fixes itself to the skin of a person in bathing or wading, and implants the ova in some superficial vein. If such be the case, it is easy to understand that the hatching process and irritation attending the movements of free embryos would result in an indolent form of ulceration, and that the little animals might be carried by the circulation from the legs to the urinary organs. It is certain that new colonists in the Cape are very apt to be attacked by indolent sores on the legs, and it seems not unlikely that their origin may thus be accounted for.

Of the *treatment* of this parasite, Dr. Harley observes: "I have found that a draught composed of ♏xv each of oil of turpentine and male fern, and ♏v of chloroform, in ℥ij of tragacanth mixture, given every morning, brought away great numbers of the ova. The saline condition of the urine is much diminished, and the renal irritation and pain due to the presence of crystalline concretions are much relieved by the administration of bicarbonate of potash in copious draughts of water. The alkali dissolves the uric acid, which I believe to be the cementing medium of the oxalic deposits, and thus the disintegration of the calculi is facilitated, and their formation prevented."

From the researches of Siebold on the trematode worms, it may be assumed, that between the ciliated embryo above mentioned and the adult sexual worm, there are two other distinct forms, which serve to complete the chain of metamorphoses connecting these two extremes of development. Fresh water mollusca and fish are probably the victims selected by the parasite during its development through these intermediate stages. Harley on these grounds suggests the following prophylactic measures in districts affected with endemic hæmaturia: 1. The water should be conveyed from its source to its destination in covered channels, so that the ova contained in the urinary and fecal products of those infested with the parasite may be prevented mixing with it. 2. Drinking water should be filtered. 3. Salads which may entangle small mollusca containing parasites, and uncooked mollusks and fish (as smoked fish), should be carefully avoided.

When the ravages of the parasite are confined to the bladder and prostate local means may be employed. Dr. Harley tried the effects of injections of wormwood, quassia, and iodide of potassium. He obtained good results only with the last. He recommends a solution of 20 or 30 grains of the iodide in 5 ounces of tepid water to be injected every second or third day.

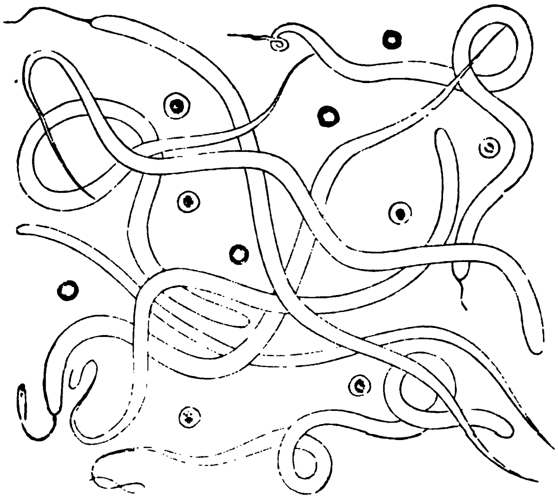
III.—FILARIA SANGUINIS HOMINIS.—*Lewis*

In 1872 Dr. T. R. Lewis made the interesting observation that, in India, chylous urine always contained large numbers of a minute nematoid worm, to which he gave the name of *Filaria Sanguinis Hominis*. Further observation led him to the discovery that the same worm was present in the blood of chyluric patients, and in such numbers that a single drop of blood extracted from the finger almost invariably showed the presence of one or several filariæ. The symptoms found coincident with this worm by Dr. Lewis were past or present chyluria, and, less frequently, persistent diarrhœa, conjunctivitis, deafness, elephantiasis of the scrotum or legs. In one case filariæ were found abundantly in the blood of a man three years after the chyluria had ceased.

The Filaria Sanguinis Hominis is a long narrow worm about the breadth of a red blood-disk and $\frac{1}{75}$ of an inch in length (*see*

Fig. 75). When examined in fresh drawn blood it exhibits very active, wriggling, snake-like movements. It consists of a delicate hyaline tubular envelope closed at both ends. Within this the worm elongates and contracts. Under a magnifying power of 600 diameters transverse striæ can be detected and granular aggregations, but no distinct evidence of oral and anal orifices, nor of any other structure. Its habitat is the blood, and, owing to its minute size, it is capable of circulating through the capillaries. Whether it is capable, like the trichina, of inducing general febrile disease is uncertain—though one of Dr. Lewis's cases indicates this pretty strongly. It local effects are supposed to depend on the formation of aggregations of filariæ which block up the capillaries, and cause by their active movements irritation and rupture of the blood channels and lymphatics, and thus lead to the appearance of chyle and blood in the urine, to discharges from the cutaneous surfaces, with thickenings and inflammation of the skin and intestinal mucous membrane—giving rise to hypertrophy (elephantiasis) of the integument, diarrhœa, and so forth.

FIG. 75.



Filaria Sanguinis Hominis—from a drop of blood obtained by pricking the finger of a European woman suffering from chyluria. A few blood-disks are introduced to show the relative size of the Filariæ. × 300. [After Lewis.]

Dr. Bancroft, of Brisbane, discovered filariæ in the blood of persons suffering from chylous urine in Queensland, Australia. He kindly sent me some specimens preserved in glycerine and water enclosed in hermetically sealed capillary tubes. I had no difficulty in identifying these with the filariæ described by Lewis. Dr. Bancroft infers from his experience that the disease is not communicated from person to person in the same family, but thinks that it may be communicated by drinking contaminated water. Dr. Bancroft found it much easier to detect the filariæ in the blood than in the urine.

The filariæ found by Dr. Bancroft were also submitted for examination to Dr. Cobbold, who identified them with Lewis's filariæ, and showed that they were only the embryo form of a worm of which the adult was as yet wanting. Dr. Bancroft, in 1877, after renewed searchings, was able to announce the discovery of the adult worm, which was styled by Dr. Cobbold *Filaria Bancrofti.* The specimens first examined were obtained from a lymphatic abscess of the arm and from a hydrocele of the spermatic cord, and were discovered to be examples of the female worm. It is about three inches in length, and at the thickest part about $\frac{1}{90}$ of an inch in diameter. A complete uterine system terminates in a vaginal pouch, opening near the head, and from the genital opening large numbers of the embryo filariæ are discharged. The discovery of the adult worm has since been confirmed by Lewis and Manson, the latter of whom saw the parasite *in situ* in a dilated lymphatic. The male worm has not yet been discovered.

The blood of a patient who is the host of the parasite, does not contain the embryo filariæ at all times of the day, but a most remarkable periodicity has been observed in their appearance. In ordinary circumstances, the filariæ make their appearance in the blood at sunset, increase in numbers up to midnight, and then gradually diminish, to disappear entirely at about nine or ten o'clock in the forenoon. Manson showed that this periodicity was interfered with by a febrile attack, but Mackenzie (*see* page 353) made the remarkable observation that the periodicity could be completely reversed by causing the patient to rest in the daytime and to be active and take his meals during the night. Under such conditions the rise and fall in the numbers of the filariæ were again observed, but at similar hours of the day and not of the night. The fate of the filariæ in the body is as yet doubtful. In Dr. Mackenzie's case the number of filariæ in the blood was estimated at from thirty-six to forty millions, at the period of the greatest number.

It is now apparent that a patient may for a long period be the host of this parasite without showing any morbid symptoms. It is evident then that some additional circumstances must occur to cause the severe symptoms mentioned above, which are probably due to obstruction of the lymphatic channels. Manson is of opinion that this event is nothing else than an abortion of the parent worm. In the early stages of its development the filarial embryo lies curled up in its capsule, but as it nears maturity, gradually elongates and stretches its capsule until it assumes the thread-like form in which it is usually observed. In this shape it is able to pass freely through the various lymphatic and hæmic capillaries. If, however, the embryo be discharged into the blood stream before the process of elongation is completed, according to Manson its more rounded shape renders its passage along the smaller lymph capillaries impossible, and the lymph stream is hence obstructed.

The researches of Manson have also revealed the interesting fact that the female of a certain species of mosquito acts as the intermediary host to the parasite. The embryo filariæ shows a tendency to curl round any fine object with which they are brought into contact. In this way, it is believed, they curl round the proboscis of the mosquito, and are so transferred to the stomach of the insect. This is rendered probable by the fact that the blood found in the stomach of the mosquito contains more filariæ than the blood of the host. Most of the filariæ embryos are either digested or passed out with the feces of the mosquito. Some, however, develop and are finally liberated by the death of the mosquito and disintegration of its body. Their future transference to man is probably by means of drinking water.

IV.—STRONGYLUS GIGAS.—*Rudolphi.*

(*Eustrongylus Gigas—Diesing.*)

This is the largest of the nematoid worms, and in its general conformation resembles a gigantic lumbricus. The male measures from ten inches to a foot in length, and a quarter of an inch in breadth, while the female has sometimes a length of more than a yard. It is distinguished from the common round worm by its reddish color (which is, however, apparently due to the sanguineous fluid in which it is usually bathed), its greater size, and the existence of six nodules or papillæ round the mouth. The Ascaris lumbricoides has only three oral papillæ.

The worm is almost peculiar to the kidney and urinary passages, and is very rarely found elsewhere. It inhabits weasels, the North American mink, and has been found in the dog, wolf, horse, ox, and some other animals; according to Schneider certain kinds of fish are the intermediate bearers. It is of extreme rarity in the human subject. Of the seventeen alleged cases collected by Davaine, he only classes seven as even probable instances. There are none of recent occurrence; and it is evident that most of the alleged cases were really examples of lumbrici, which had penetrated into the urinary passages from the intestines.

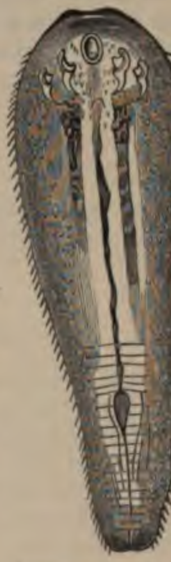
A very fine specimen is preserved in the museum of the London College of Surgeons, which I have had an opportunity of examining. It is an undoubted strongylus, more than a foot long. It originally belonged to Brookes's museum, and is entered in Brookes's catalogue as "an uncommonly fine specimen of an enormous worm (strongylus gigas) found in the kidney of a patient of the late Thomas Sheldon, Esq."

V.—PENTASTOMA DENTICULATUM.—*Rudolphi.*

This is a very minute encysted parasite, about a line and a half long, club-shaped, with a double pair of hooks, and devoid of sexual organs (*see* Fig. 76). It is conjectured by Davaine to be the larva of pentastoma tænioides, which infests the frontal sinuses of dogs and horses. No symptoms are known to be produced by it.

The only known instance in which the parasite was found in the urinary organs is the following: In making the autopsy of a painter, sixty-two years of age, who died of Bright's disease, Wagner found on the convex border of the right kidney a small, whitish, slightly raised oval patch of fibrous appearance, about one-seventh of an inch long. It was situated under the capsule of the kidney. This little body was hollow in the interior: it contained a yellowish mass, which on examination disclosed the presence of a worm, which was recognized as the pentastoma denticulatum of Rudolphi.

FIG. 76.



Pentastoma denticulatum, greatly magnified. [After Zenker.]

This worm is common on the surface of the liver in goats, oxen, rabbits, cats, and some other animals. It has also recently been found on the surface of the liver in man, by Zenker in Dresden, Heschl in Vienna, and by Virchow, Wagner, and Frerichs in other parts of Germany. Cobbold states that Dr. Murchison, during the time he held the office of Pathologist at the Middlesex Hospital, diligently searched for it without success.

VI.—ERRATIC WORMS.

Intestinal worms sometimes penetrate into the urinary passages, and are voided with the urine. In women thread worms occasionally creep into the bladder through the urethra; and in both sexes lumbrici, and joints of tapeworm, have been known to creep into the bladder through fistulous communications caused by abscesses, passage of pins, lithotomy, etc.

VII.—SPURIOUS WORMS.

The *spiroptera hominis* of Rudolphi, the *diplosoma crenata* of Farre, and the *dactylius aculeatus* of Curling, have been clearly proved by Schneider and Cobbold to be examples of imposition —witting or unwitting on the part of patients. The history of the so-called diplosoma crenata of Farre furnishes one of the most remarkable examples ever put on record of long-continued and successful deception practised on scientific inquirers. The following references may be consulted on the subject: W. Lawrence, "Med.-Chir. Trans.," vol. ii. 385; A. Farre, "Beale's Archives of Medicine," vol. i. p. 290; A. Schneider, "Reichert and Dubois's Archiv," 1862, p. 275; Cobbold, "Entozoa," pp. 403, 409; Curling, "Med.-Chir. Trans.," vol. xxii. p. 274.

CHAPTER XIV.

ANOMALIES OF POSITION, FORM, AND NUMBER OF THE KIDNEYS.

THE kidneys are subject, like other organs, to certain deviations from their natural situation, form, and number. Most of these deviations are congenital; others are acquired later in life through accident or disease. Some of them are appreciable during life, and are liable to be confounded with wholly different pathological states; others are entirely latent, and are, so long as the healthy state is maintained, nowise detrimental to the subject of them, but bring greatly increased risks, under certain contingencies of obstruction to the course of the urine.

I.—ANOMALIES OF POSITION.

The kidneys may occupy an unnatural situation, and remain permanently *fixed* in that situation; or the misplaced organs may possess a certain *mobility*.

A. Fixed Malpositions of the Kidneys.

The kidney may be displaced downwards, upwards, or laterally, by the pressure of a tumor growing in its vicinity, or by an enlarged liver, spleen, pancreas, or suprarenal body; in these cases the malposition is *acquired*. But the malposition may also be *congenital*. Instead of lying beside the vertebral column, deep in the lumbar region, the organ may be fixed in front of the vertebræ, or on the brim of the pelvis or within that cavity; in a case figured by Ruysch, the kidney lay crosswise, with its hilus turned upwards, the ureter descending behind it.

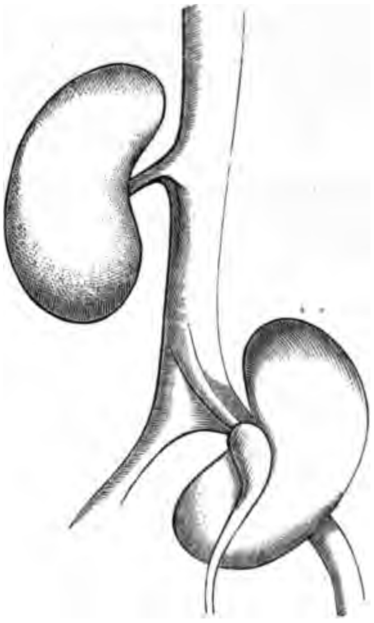
A kidney congenitally misplaced usually deviates more or less from its natural configuration, and is associated with malposition of some portion of the large intestine and peritoneum. The renal artery and ureter also necessarily deviate less or more from their natural distribution. The corresponding suprarenal capsule does not (in congenital cases) follow the kidney into its abnormal situation, but invariably occupies its usual place in the lumbar region.

By far the most common, and also the most practically important, of the fixed misplacements of the kidney, are those in which the organ lies within or upon the brim of the pelvis. In these cases the misplaced organ is liable to be felt during life, either through the abdominal wall or the vagina, and to be mistaken for some other object; if it lie within the pelvis it may embarrass and complicate parturition.

In twenty-one cases of congenital malposition of the kidney which I have been able to collect and compare, the abnormality was, in every instance, confined to one kidney, and the left kidney was much more commonly affected than the right (left 15, right 6).

The most frequent of these deviations was to find the kidney lying obliquely on the sacro-iliac synchondrosis, as represented in Fig. 77. In some of the cases, the organ was fixed beside the uterus, or transversely between the rectum and bladder, or across the prominence of the sacrum.

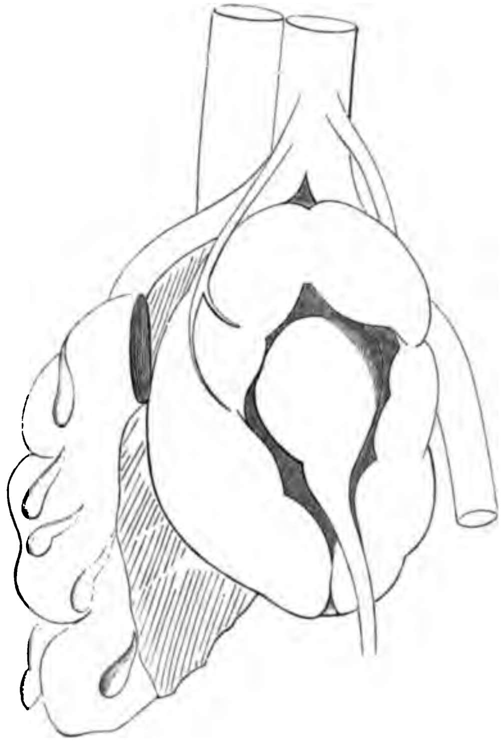
Fig. 77.



Left kidney, lying on the left sacro-iliac synchondrosis. (From a drawing in the possession of Dr. Renaud.)

Mr. Canton has described and figured a curious specimen, taken from a man who died (of bronchitis) at the age of twenty-seven. There were no renal symptoms during life. The right kidney was in all respects normal; but the left was situated below, and between, the bifurcation of the aorta, as shown in Fig. 78. Instead of presenting the ordinary kidney-shape, the gland was rudely oval, and, on some parts of its surface, lobulated. The pelvis of the organ was directed almost immediately forward, and the upper portion of the ureter was dilated, owing to the impaction in it of an oxalate of lime calculus, weighing 2½ drachms. The left renal arteries were two in number, and sprang from the forepart of the aorta at a short distance above its division. The sigmoid flexure of the colon was placed, as represented in the engraving, on the right side of the kidney.

FIG. 78.



Mr Canton's case of misplaced and lobulated kidney. (From the the Transactions of the Pathological Society, vol. xiii. p. 147.)

It rarely happens, that malpositions of this class produce any evidence of their existence during life; but sometimes, as in the two following cases, the misplaced organ forms a palpable tumor in the abdomen, which is liable to be mistaken for something of a more serious character; or, in the female, it may constitute an obstacle to parturition.

CASE 1. *Left kidney malformed, and situated over the left sacro-iliac synchondrosis, mistaken for an abdominal tumor* (Durham, "Guy's Hosp. Reports," 1860, p. 407).—Mr. W. S., previously in good health, suffered, at the age of forty-five, from a severe attack of fever. During his recovery, he noticed, for the first time, a tumor deeply seated in the hypogastric region, somewhat on the left of the middle line. This tumor was found on examination to be oval in form, somewhat elastic to the touch, and fixed. It was not nodulated, nor did it present any distinctive elevations or depressions. Manipulation gave rise to very disagreeable sensations, but not to acute pain. Considerable alarm was felt by the patient, especially as some members of his family had died from "tumor in the abdomen." In the course of a short time, when convalescence from the fever was established, a second opinion was taken. The conclusion arrived at was, that there existed in the lower part of the abdomen "a tumor of doubtful character." Iodine ointment was applied, and iodide of potassium taken internally. The treatment was continued for some time, but, of course, did not produce the slightest effect on the tumor. Mr. S. never thoroughly recovered his health and strength, and about four or five years after his attack of fever, died of pulmonary disease.

Autopsy.—Upon opening the abdomen, it was at once seen that the supposed tumor was nothing more than the left kidney, which was situated over the sacro-iliac synchondrosis, and extended somewhat on to the promontory of the sacrum, and also, by its lower part, into the true pelvis. The colon formed no sigmoid flexure in the left iliac fossa, but passed across the middle line; and the commencement of the rectum was on the right side of the sacrum. The suprarenal capsule was in its normal position. The kidney presented two depressions, which divided its surface somewhat indistinctly into three portions. The principal arterial supply was derived directly from the aorta, by a branch coming off just above the bifurcation; a branch of the common iliac artery of the *opposite* side, and a branch of the internal iliac of the same side, also supplied different parts of the organ. There was one principal vein, which passed from the internal and posterior part of the kidney into the vena cava just above the junction of the common iliac veins. The ureter resulted from the junction of four branches; of these, two came from the upper and posterior part, while the two principal ones came from the anterior and lower part; these branches joined one another about an inch from their several points of exit from the organ. Thus this kidney presented no distinct hilus, nor, consequently, did it possess the characteristic kidney-shape. The right kidney was in its natural position, and both glands were quite healthy.

CASE 2. *A misplaced left kidney offering an obstacle to parturition* (Hohl, cited by Rayer, loc. cit., tom. iii. p. 774).—The subject of this observation was a woman, in whom the left kidney was situated deeply on the inside of the psoas muscle. In two labors, through which this woman had passed, a tumor was formed each time on the left side of the pelvis, which excited fixed and increasing pain with each contraction of the uterus; the passage of the head was thereby retarded, but both accouchements were happily accomplished.

THE DIAGNOSIS of a misplaced kidney, forming a pelvic or abdominal tumor, rests on the moderate size and the smooth elastic feel of the tumor, together with the existence of a want of fulness, or a slight hollowing, of the corresponding lumbar region—denoting the absence of the kidney from its usual place. The shape of the tumor when reniform, of course greatly assists the diagnosis; but in a large majority of such malpositions, the peculiar kidney-shape is not preserved.[1]

B. Movable Kidneys.

Vague allusions to mobility of the kidneys are found in the works of the old writers (Mesué and Riolan), but to Rayer belongs the credit of having first pointed out the practical bearing of this condition, and the symptoms and signs by which it may be recognized during life. In this country the subject has been ably illustrated by Dr. Hare. Mr. Durham has brought together and collated all the instances (10 in number) which, up to that time (1860), had been verified by post-mortem examinations. Oppolzer and Henoch, in Germany, have contributed a number of cases; Fritz has analyzed all the cases published prior to 1859. Since that date comprehensive essays on the subject have been contributed by Becquet and Rollet, and still more recently by Landau. The following account is based on an analysis of 70 cases, partly derived from the sources above indicated, and partly contributed by myself.

PHYSICAL SIGNS AND SYMPTOMS.—The kidneys, in their normal state, are secured in their position by a thick investment of adipose tissue, and a reflection of the peritoneum which passes over their anterior surfaces; but under certain circumstances, one or both kidneys break away from these not very firm attachments, and float loose amid the abdominal viscera—no longer bound except by their bloodvessels and excretory ducts. The degree of mobility and of change of position which the kidney acquires in these cases varies greatly. In the generality of cases, the organ descends, when the patient is standing upright, below the margin of the ribs, and occupies a diagonal position, extending from below upwards and outwards, midway between the costal border and the umbilicus. In this situation it forms an oblong tumor, having the shape and feel of the kidney. It can be pushed in various directions—upwards, or downwards, or laterally—over a space of several square inches. In persons with flaccid bellies, the gland can be actually grasped with the hand; and a sickening, sinking sensation is experienced when it is compressed; otherwise it is usually painless. When the patient lies horizontally, the displaced kidney can be thrust back again by the hand, into its natural situation in the lumbar region; but it generally resumes its unnatural place when the pressure is withdrawn. The respiratory movements and the posture of the body exercise a marked influence on the position of a movable kidney. Deep inspiration causes it to descend, and deep expiration to ascend; it falls over to the linea alba, or in the opposite direction, as the body is inclined to this or that side. In the slighter cases, half or three-quarters of the length of the organ is palpable through the soft abdominal walls, along the borders of the false ribs: but the displacement is generally more considerable than this; in a case mentioned by Johnson the kidney had drifted below the umbilicus; in another, related by Day, the kidney lay in the iliac fossa, and could be moved hither and thither over a space of three or four inches. When the patient reclines, the displaced organ occupies a higher position than after long standing or walking. Percussion does not yield a dull sound over a movable kidney, but a muffled tympanitic note. When the loins are examined, a flattening or slight hollowing of the renal region, on the side of the displacement, is perceived; and the percussion note is tympanitic—showing that the absent kidney is replaced by intestine. When the organ is thrust back by the hand into its original position, the natural bulging in the loin is restored, and the bowel sound disappears.

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[1] For three additional cases of fixed malposition of the kidney, *see* Hausmann, Monatsch. f. Geburtsk., xxxiii. p. 401; Gosselin, L'Union Méd., 1869, 115; and Powell, Lancet, 1882, i. p. 1033.

The subjective symptoms vary a good deal. In some cases the symptoms are so slight as not to attract the patient's attention, and the anomaly is only detected by an accidental examination of the abdomen in the course of some other complaint. There is, however, usually marked suffering and inconvenience. The most common symptoms are a dragging pain in the side aggravated by walking or standing. Sometimes the patients are conscious of the existence of a movable tumor in the abdomen, which gives them serious uneasiness, and produces a hypochondriacal and depressed state of mind. In one instance the movements were mistaken for those of a child in the womb. Sometimes the stomach is disturbed[1]—more frequently the bowels are affected, either with fitful diarrhœa or with constipation. Pains of a neuralgic character are generally experienced in the neighborhood of the displaced organ, and radiate thence in various directions—into the loins, round the waist, down to the lower parts of the belly, and along the thighs.

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[1] Digestive disturbances have been attributed to the traction which a movable kidney on the right side may exert on the second portion of the duodenum. Obstruction to the intestine, so produced, has been said to have caused dilatation of the stomach. (Müller-Warneck, Berl. klin. Wochenschr., 1879, No. 30; Stiller, Wien. Med. Wochenschr., 1879, Nos. 4 and 5.)

In some cases severe paroxysms resembling biliary or nephritic colic occur from time to time, accompanied with sickness, vomiting, shivering, faintness, and signs of local peritonitis. During these attacks the kidney becomes considerably swollen, and forms an immovable and painful tumor in the abdomen. When the paroxysm has subsided—which it usually does after a few days of rest in bed and the use of opiates and warm external applications—the kidney becomes again reduced in size and resumes its mobility. Sometimes, however, it contracts inflammatory adhesions to the parts around, and remains permanently fixed in its new position. The exact cause of these paroxysms is somewhat obscure. They sometimes set in suddenly, while the patient is in bed, without appreciable cause; more often they follow some unusual exercise, or an indigestible meal, or they occur at the menstrual periods. In some cases, at least, it is probable, as Gilewski has pointed out, that they depend on a sort of strangulation of the kidney from the pressure of the displaced organ on its own ureter, and a consequent obstruction to the flow of urine, followed by acute engorgement of the kidney with dilatation of the pelvis and pyelitis. Such an explanation would account for the fact that the urine sometimes becomes bloody and purulent for a time after one of these attacks (Rollet, l. c., p. 20).

The secretion of urine generally goes on without any alteration; but, occasionally, micturition is unnaturally frequent, and accompanied with more or less pain. Among other complications which have been observed in cases of movable kidney may be mentioned epigastric pulsation, the discharge of uric acid gravel, the occurrence of hydronephrosis, Bright's disease, œdema of the lower limbs from compression of the ascending cava by the displaced kidney, and obstinate constipation from similar compression of the colon.

The following examples furnish typical illustrations of the physical signs and of the varying symptoms associated with movable kidneys.

CASE 1.—On the 19th of February, 1867, I saw at the Crumpsall Workhouse, with Dr. Simpson, a woman named E. Canning. She was 31 years of age, and was suffering from chronic phthisis. She had had two children, the younger being two years old; had never had any blow or fall, and menstruation had always been regular and painless. About three weeks previously, she discovered a "lump" in her right side after an attack of diarrhœa, and called the attention of Dr. Clarke, the resident medical officer, to it. She had never been very stout, but was now considerably emaciated.

The right kidney was very movable; as the patient lay on her back, it occupied the position indicated by the continuous line in Fig. 79, where a smooth, slippery, globular object was felt, which was evidently the lower end of the right kidney. By thrusting the thumb into the loin, the tumor was pushed forward and came within complete reach of the hand, so that it could be partially grasped, and thrust back into the loin, or moved about in a circle represented by the dotted line. It was felt to be oblong, with rounded smooth ends. The left kidney was also slightly movable; it did not come forward towards the umbilicus, but when the patient sat up, its lower end could be felt in the flank, an inch below its natural situation, and it could be pushed up until it disappeared beneath the ribs.

FIG. 79.

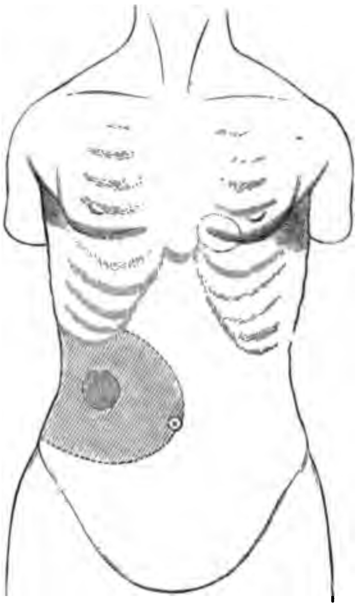

Diagram showing the area over which the right kidney could be moved in the case of E. Canning.

The patient suffered no inconvenience whatever from the state of the kidneys; but as she was constantly in bed, she was scarcely in a condition to suffer any. Two months after I first saw her she died, and the body was examined by myself, with Dr. Simpson and Dr. Clarke.

The case was one of ordinary pulmonary tuberculosis.

The kidneys were found quite healthy; the right lay loose in the right hypochondrium, projecting an inch and a half below the margin of the liver; its vessels and excretory duct formed its sole attachments. There was scarcely any fat about it; but the general emaciation was so great that this did not appear singular. There was no abnormal distribution of the peritoneum, nor of the bloodvessels. The left kidney lay almost in its normal position, but about an inch lower. The artery and vein of the right kidney were half an inch longer than those of the left.

CASE 2.—Mrs. D., æt. 36, the mother of several children, had been occasionally under Dr. Hare's care for several years. She had suffered from anæmia and oligo-menorrhœa, but got quite well of these. She had afterwards an attack of gastrodynia, when she had also much languor and debility, with weight and sinking sensation at the epigastrium.

In the spring of 1852, Dr. Hare attended her for a slight bronchial attack; when she got better of that she complained of a "beating sensation" down the middle of the abdomen, and also of having at the upper part of it, on each side "some swellings which on pressure slipped up under the ribs." She had had a sinking sensation at the epigastrium for years, but it was only about twelve months that she had felt, on applying her hand there, a tumor in one (the right?) hypochondrium, and about four weeks another on the other side. The aortic impulse had been troublesome for five weeks past.

FIG. 80.

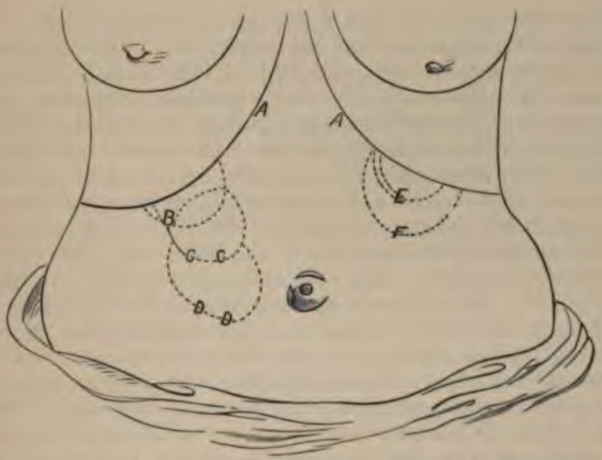

Diagram showing the varying positions of the kidneys in the case of Mrs. D. A, A. Margins of costal cartilages. B. Right kidney; ordinary position when patient is in recumbent position. C C. Ditto, on deep inspiration. D D. Ditto, position to which it can be moved. E, F. Left kidney; changes in position of. [After Hare.]

On making an examination of the abdomen (which was rather thin, and of short antero-posterior diameter, while the parietes were also flaccid), the aortic impulse was found to extend from the upper part of the epigastrium to more than an inch below the umbilicus, and it was exceedingly well marked and strong. The left kidney was situated lower than usual, but readily glided when pressed upon, from under the fingers, deep into the hypochondriac region, while, on the other hand, it might be pushed some distance downwards; the right kidney presented the same phenomena, except that it was much more mobile, and could be detruded downwards so far, that the whole of it could be felt some distance below the costal cartilages, and its form well made out, owing to the thinness of the parietes (*see* Fig. 80).

A belladonna plaster was applied to the abdomen, and some tinct. ferri sesquichlor. and tinct. calumbæ were given. The patient was afterwards seen several times by Dr. Hare, and again very recently; at times she had been free from all renal pain, but lately she had again felt somewhat weaker, and she had had more, both of the abdominal impulse and of the dragging sensation in the loins, though the pain was by no means so much there as it was a few years ago. On examining the abdomen, the mobility of the kidneys (especially of the left one) appeared to be less than formerly, though that of the right one was still very notable (Hare, "Med. Times and Gaz.," 1858, i. p. 86).

CASE 3.—On July 22, 1869, I was asked to see, with my colleague, Dr. Morgan, Mrs. P., a married lady, aged 27. She was a spare, anæmic, but active person. She had had six children in little less than seven years. Previous to her marriage she was plump and rosy. For several years she had suffered from profuse leucorrhœa; but with the exception of occasional dyspepsia, she had enjoyed fair health until Christmas, 1867, when she was suddenly seized while in bed with a violent attack of shivering, accompanied with retching and sickness and intense pain in the situation of the gall-bladder. This attack lasted 48 hours, and gradually subsided under the influence of morphia and hot fomentations. From this time (Christmas, 1867) until the date of my visit (July, 1869) Mrs. P. was frequently subject to a repetition of similar attacks, which occurred at longer or shorter intervals. At one time, during many consecutive months, the seizures came on every fortnight. Any unusual exercise or excitement was sufficient to provoke them. About a month before my seeing the patient, she went to Buxton and took the baths. While there she was seized with one of her old attacks, but of greater severity than any previous ones. This attack lasted for upwards of a week, and one morning the patient discovered a swelling or tumor in the abdomen, to which she directed the attention of Mr. Shipton, who was in attendance on her. The swelling felt like the end of a large cucumber, and extended downwards from the situation of the gall-bladder to within two inches of Poupart's ligament. It was painful on pressure, and it had made its appearance somewhat suddenly—for no tumor of the sort could be detected when the abdomen was examined five days previously. Mr. Shipton considered it to be the displaced right kidney. When the patient was examined by me the tumor was much smaller, and painless. It projected from beneath the margin of the liver to the extent of about two inches. It was evidently adherent to the surrounding parts, and could only be moved within a limited area. It had a smooth rounded outline, and appeared about the size and shape of the lower end of the kidney. It could not be pushed back into the flank. The site of the right kidney felt flattened and empty as compared to that of the left kidney. I had no doubt that the tumor was the displaced right kidney, and that when first perceived by Mr. Shipton it was much swollen and congested. It appeared probable that during the attack at Buxton the organ had contracted inflammatory adhesions to the surrounding parts, and was now permanently fixed in its new position. The patient was fitted with an abdominal bandage, and was directed to avoid all violent exercise.

From a communication I received from Dr. Morgan on April 15, 1872, I learn that Mrs. T. has suffered very little from the tumor since the date of our visit in 1869. She wore the abdominal belt for about two years, and has now left it off. The only time she suffers much discomfort in the region of her erratic kidney is at the menstrual periods. For some two days before the catamenia appear the kidney seems to become larger, is sensitive to the touch, and at times somewhat painful even without being touched. Walking exercise does not suit her, and, if prolonged, is apt to cause discomfort in the kidney; but she can ride on horseback or dance without inconvenience. The kidney still occupies much the same situation as in 1869.

CASE 4.—Mr. D., æt. 45, consulted me in July, 1870. He stated that two years previously he had fallen on the ice on his left side, and that ever since he had suffered from dragging-pains in the right loin and the neighborhood of the ascending colon, with irritable bowels and loss of flesh. Although moderate exercise caused him no great inconvenience, the pains always disappeared entirely in bed. Two or three loose motions of the bowels occurred daily. Various methods of treatment had been tried without any relief.

On examining the abdomen, I found an oval tumor about the size and shape of the kidney, lying on the edge of the pelvis, in the vicinity of the cæcum. On deep pressure it slipped upwards from under the fingers; very little pain accompanied this manœuvre. When the loins were examined, a conspicuous flatness was perceptible in the right renal region. There was no doubt that the right kidney had descended about its own length, and lay in the iliac fossa. He was directed to push up the tumor while in bed, and to apply a tight band round the waist, with a pad beneath it, over the position of the displaced organ.

The effect of the treatment was immediate; the diarrhœa entirely ceased, and the dragging pains scarcely troubled him. I saw this gentleman several times subsequently; he had had a leathern girdle with a pad made for himself, which kept the kidney approximately in its natural position; and so long as he wore this girdle the bowels remained quiescent and the pains were insignificant; but if he left it off, the old symptoms returned forthwith.

I am indebted to the late Dr. Ritchie for the notes of the two following cases. In one of them the kidney became movable after a fall on the loin; the other illustrates the occurrence of congestion and enlargement of the kidney at the menstrual periods.

CASE 5.—Mr. J., æt. 25, came under my care on 24th July, 1871. He informed me, that about a fortnight previous to my seeing him, he had been running hurriedly downstairs, when his foot slipped off the edge of one of the steps, and he fell backwards, receiving the full force of the blow on his right loin. Between the accident and the time of my seeing him he had been in constant suffering, and by the advice of a medical man, who looked on the case as one of "lumbago," had assiduously employed friction with various liniments, with the effect of aggravating and not alleviating his pain. He was apparently in robust health, with the exception of the ailment for which he sought my advice. He complained of a constant dragging sensation in the belly, which, after standing or walking, became developed into a severe pain darting between the right loin and the belly. On two occasions, after prolonged standing, he had also pain darting down the right thigh, causing him so much suffering that he was unable to attend to his business. On examining the abdomen a hard smooth tumor was found in the right hypochondriac region. It was quite distinct from the liver, was freely movable, and had an irregularly rounded outline. Handling the tumor gave rise to no positive pain, but to sickening sensations. When the patient lay on his back the tumor could not be discerned unless he took a deep inspiration; it then appeared to pass from under the ribs, reascending with deep expiration. It could be most readily seen as the patient lay on his back with a slight inclination to the left side. Percussion over the tumor elicited a slight amount of dulness. There was considerable pulsation in the abdominal aorta, extending from the epigastrium to about an inch below the umbilicus. The patient stated that he had not noticed this previous to his accident, and that it became troublesome if his bowels were constipated. On examining the lumbar regions, the right was seen to be slightly flattened or hollowed out, as compared with the left; and while, on percussing the left lumbar region, the normal renal dulness and resistance were encountered, the right was almost perfectly tympanitic. There were no urinary symptoms. A broad flannel bandage was firmly fastened round the patient's abdomen, upon which he expressed his relief from the dragging which he previously complained of. This, with careful regulation of the bowels, appeared to be all the treatment indicated, for on a subsequent visit, a week after first seeing him, he stated that he had remained perfectly free from pain or discomfort since the application of the bandage.

CASE 6.—M. K., æt. 17, came under my care as an out-patient of the Hulme Dispensary on 8th August, 1871. She was thin and anæmic; menstruation regular but profuse. She stated that she had enjoyed moderately good health till about six weeks prior to my seeing her, when she noticed a swelling in the abdomen on the right side, a little below the ribs, and a "great fluttering" at the pit of the stomach. Since then the bowels had been very irregular, being sometimes constipated and sometimes very loose. The fluttering was worst when the bowels were confined, and during her menstrual period, when she thought the swelling was larger. She stated that she had never been in the habit of tight lacing, and no account of any fall, blow, or extreme exertion could be elicited which could be construed into a probable cause of the swelling. On examining the patient in the erect position, a smooth hard swelling, apparently about the size of the clenched fist, was detected just below the ribs on the right side. Its lower margin reached to within an inch and a half of the anterior superior iliac spine; it was movable to a greater or less extent in every direction within the abdominal cavity, but the greatest mobility was in the direction of the normal site of the kidney, viz., upwards and backwards beneath the ribs. When the patient lay on her back, the lower edge of the tumor could just be seen below the ribs; it could be seen to pass much lower on her taking a deep inspiration. The tumor was sensitive to pressure, and the examination gave rise to nausea, which the patient afterwards told me lasted for nearly two hours. There was no great difference either in appearance or on percussion between the right and left lumbar regions. There was strong pulsation in the abdominal aorta, which could be felt through the thin parietes to be slightly on the right of the median line. The heart and lungs were apparently healthy; there were no urinary symptoms except a slight smarting on micturition, which came on synchronously with a vaginal (gonorrhœal?) discharge a few days previously. The patient remained under observation for ten weeks, and a broad flannel bandage was firmly applied to the abdomen. After the cessation of the discharge and smarting, she was put on cod-liver oil and an iron tonic. An opportunity was afforded of watching the tumor during two menstrual periods—and at that time its size was seen to be increased by fully one-half—it was much more sensitive to the touch, and the pulsation of the abdominal aorta was considerably stronger than during the intervals.

Etiology.—Mobility of the kidneys is much more common in women than in men; and more common on the right side than the left. Of the seventy cases which I have collated, sixty-one were women and only nine men. In sixty-five cases, information is given as to the side affected:

In 42 the right kidney alone was movable.
In 9 the left kidney alone was movable.
In 14 both kidneys were movable.

The age of the patients varied from sixteen to sixty-five years —the general range being between twenty-five and forty, which corresponds roughly to the child-bearing period in women.

Mobility of the kidneys, judging by my own experience, is much more frequent than is usually supposed; and many cases of inexplicable pain in the abdomen and intestinal disturbance are due to this cause. A large number of cases are undoubtedly overlooked. Rollet states that of 5500 patients admitted into Oppolzer's Clinique, and examined carefully as to this point, 22 had movable kidneys—a proportion equal to 1 in 250.

In a certain number of cases no clear determining cause can be discovered; but, as a rule, the antecedent history of the patient discloses some circumstance or circumstances to which the anomaly can be traced. In many instances the affection is due to repeated or difficult labors; and this is one reason of the greater frequency of movable kidneys in women than in men. The alternate tension and relaxation of the abdomen occurring in successive pregnancies, and the convulsive muscular efforts which accompany parturition, must evidently tend to loosen the attachments which hold the kidney in its place, and favor its migration, under the force of gravity, into a lower position in the abdomen.

The disproportionate frequency of mobility of the kidneys in the female sex, and especially on the right side, is also partly due to tight lacing. In reference to this point, Cruveilhier observes: "I have often observed, in women who wore tight stays, the right kidney to lie sometimes in the right iliac fossa, sometimes in front of the sacro-iliac synchondrosis, sometimes even in front of the vertebral column, at the level of the adherent border of the mesentery, in the substance of which it was placed. The kidney, thus accidentally displaced, enjoys a certain mobility. This displacement of the kidney arises, when the pressure exercised on the liver by the stays dislodges the right kidney from the kind of niche which it occupies on the under surface of this organ.

"If the left kidney is not so frequently displaced as the right, that is owing to the fact that the left hypochondrium, occupied by the spleen and the great end of the stomach, bears the pressure of the stays with much more impunity than the right." (Cruveilhier, "Descriptive Anat.," vol. iii.)

Rapid emaciation in obese persons, and the removal of the capsule of adipose tissue which naturally invests the kidney, seem, in some instances, to have favored or determined the mobility of the organ. Oppolzer states, that in the cases which he had an opportunity of examining—the patients dying of some other disease—there had always been observable a deficiency of the cushion of fat about the kidney. In a case dissected by Mr. J. Adams, "the only peculiarity remarkable was, that the kidney appeared bound down in its situation more loosely than usual, and the old lady, from having been very fat, had become somewhat thinner, and her integuments appeared very lax throughout." ("Med. Times and Gazette," 1857, i. p. 651.)

In a considerable number of cases, especially in men, displacement and mobility of the kidney is produced by a blow on the loin, a sudden fall or jump, violent running, dancing, riding, or severe muscular effort or succussion of the body. These causes may produce their effect suddenly, or more slowly by frequent repetition.

Rayer mentions a case in which it appeared probable, that the kidney was dragged down, or at least left free to descend from its own weight, in consequence of displacement of the peritoneum, from a hernia of the cæcum.

Becquet has propounded a somewhat novel theory for the production of movable kidneys in women. In the cases encountered by him, there was a striking coincidence of time, between the displacement of the kidney and the menstrual period; and he was led to believe, that the kidney became congested and tumefied at these periods, and that displacement was the consequence of its increased volume and weight. He thus explains himself: "On the breaking forth of the menstrual flux, the kidneys are associated in the congestion of the generative organs, and become swelled. This fact, less rare doubtless than is usually supposed, perhaps even physiological, does it not explain the renal pain so often felt at the menstrual periods, especially in women who are subject to dysmenorrhagia?

"Thus swelled and rendered heavier, the kidney, and especially the right kidney, strains the feeble attachments which retain it, and tends to start out of its place. Soon the congestion subsides, and the organ returns to its original position; a second congestion displaces it further, and a third further still, the kidney becoming each time heavier from the incompleteness of the resolution, comes to occupy a lower position; and thus gradually, and at length, but not without suffering, breaks loose, and floats in the abdominal cavity." ("Arch. Gén.," 1865, i. p. 21.)

But although, in a majority of the cases, mobility of the kidney appears to have been acquired from some accident or circumstance arising in the course of life, there are instances in which a congenital anomaly in the anatomical connections of the gland has evidently operated to favor its production.

The committee appointed by the Pathological Society to inquire into this condition recommends that two varieties be distinguished—*movable* kidneys, where the unnatural mobility takes place entirely behind the peritoneum, and *floating* kidneys, where the peritoneum is reflected over the posterior, as well as the anterior surface of the kidney, so as to enclose it within a fold of peritoneum or meso-nephron, which permits the organ very considerable motion in the abdomen. The two conditious cannot be distinguished during life. Mr. Durham examined the body of a woman aged 34, in which the left kidney was very movable. He found the descending colon much nearer the middle line than usual, and instead of forming the sigmoid flexure in the left iliac fossa, it turned across the lumbar vertebræ, and passed down into the pelvis on the right side of the sacrum. It was manifest that the mobility of the kidney in this instance depended, in great measure, on the abnormal arrangement of the peritoneum, necessarily associated with the malposition of the colon. So far, therefore, it must be regarded as congenital. When traced from the side of the vertebral column, the peritoneum, instead of passing over the anterior surface of the kidney, only just touched the lower part of its inner border, and then, having formed the descending mesocolon, again touched its outer border. The lesser sac of the peritoneum also, instead of being confined to its ordinary limits, passed so far to the left as to cover the posterior surface of the spleen, and so far downwards as to touch, and be reflected from the upper border of the kidney. Thus there was no distinct meso-nephron, but the kidney, instead of being supported and kept down by a single layer of peritoneum, was left free to move between and beneath three diverging layers. Upon dissection it was further found that there was scarcely any fat in the lumbar region, but a quantity of very loose areolar tissue. ("Guy's Hosp. Rep.," 1860.)

THE DIAGNOSIS of movable kidney is chiefly important, from the risk of confounding an ailment which is comparatively trifling with some graver disease. In a considerable number of instances, the affection was long mistaken by the patient and his medical attendants for a tumor within the abdomen, and the patient was subjected, in addition to the alarm which such a notion necessarily engendered, to heroic and exhausting or troublesome plans of treatment. The diagnosis is indeed generally easy, and the errors committed have arisen from the possibility of this condition not having been present to the mind of the practitioner, rather than from the inherent obscurity of the case.

A movable tumor having the size and shape of the kidney, or approaching thereto, is found on either side of the abdomen, generally in the hypochondriac region; it can be pushed into the lumbar space, and again out of it, at will, by the thumb and fingers. When the corresponding loin is examined, the absence of the kidney from its usual place is rendered evident, by the flattening or hollowing of the part, and by the tympanitic note yielded on percussion. It is only in obese individuals, and in cases where the displacement and mobility are slight that any difficulty can arise. It must be remembered that the displaced organ sometimes contracts adhesions in its new position, and thereby loses, partly or wholly, its freedom of movement.

TREATMENT.—Persons with movable kidneys do not always suffer serious inconvenience therefrom, and the affection may persist without much change for an indefinite period. But, as a rule, unpleasant or painful symptoms are produced, which require to be attended to.

The most evident indication is to replace the organ in its natural position, and to keep it there. If the kidney have contracted adhesions which prevent it from reassuming its natural site, efforts must be made to support it steadily in its new place, and prevent it from sinking down by its own weight, and painfully dragging the new attachments which it has formed.[1] These objects are attained by applying a tight bandage or belt round the abdomen. This should be put on while the patient is in the recumbent position, and after the kidney (if this be possible) has been pushed back into its proper place in the loin. Sometimes a pad fitting on to the place where the organ is accustomed to protrude gives greater effect to the belt or bandage. Patients cannot always bear these mechanical appliances; but in many cases partial or complete relief is afforded by them.

Keppler[2] considered the condition so dangerous as to justify the removal of the organ. His opinion, however, has not been supported by others. Dr. Newman has recommended a much less dangerous operation, nephrorrhaphy, in which the kidney is stitched to the posterior abdominal wall. The operation has been performed seven times abroad and once by Dr. Newman, in every case with good results. Further details will be found in Dr. Newman's thesis (On Malpositions of the Kidney, "Glasgow Medical Journal," Aug. 1883).

If there be anæmia or other disorder of the general health, the removal of this by appropriate remedies is of course to be attempted. Restoration of the tone of the abdominal muscles, which, in most cases, are relaxed and flaccid, is probably the most effective means of reducing to a minimum the inconveniences which attend on mobility of the kidneys. To this end, ferruginous and other tonics, and shower baths, with avoidance of fatiguing exercise, seem to be the means best adapted. A curious case is recorded by Dr. Hare, in which the mobility of the kidneys was markedly diminished after two pregnancies; the steady pressure of the gravid uterus having apparently acted as a mechanical support to the dislodged organs.

The regulation of the bowels is a point to be carefully attended to. Accumulation of fecal matter in the large intestines invariably aggravates the inconveniences of movable kidneys. Tight lacing and all violent modes of exercise (equitation, dancing) should of course be strictly forbidden.

When the symptoms of so-called strangulation of the kidney occur—violent pains, sickness, frequent micturition, enlargement and excessive tenderness of the tumor—complete repose, in the recumbent posture, should be prescribed during the attack; hot poultices, or even leeches, should be applied over the seat of pain, and morphia administered internally.

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[1] In cases of recently formed adhesions, when the diagnosis is undoubted, Rollet states that the kidney may be pushed into its normal position by force, and the affection be at once and permanently cured. This seems a somewhat hazardous proceeding.

[2] Archiv. f. klin Chirurg., Bd. 23, S. 520.

II.—ANOMALIES OF FORM.

Deviations from the normal shape of the kidneys may exist congenitally, or be produced in after-life by the pressure of tumors or of enlargements of the neighboring organs. Some of these malformations have been already noticed, in treating of fixed misplacements of the kidney.

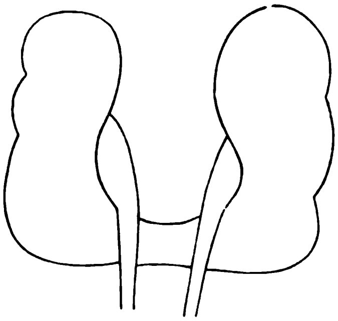
The lobulated character of the gland, which is natural to it in the fœtal state, sometimes persists more or less throughout life. Sometimes one kidney is twice or thrice as large as its fellow, although both may be perfectly healthy—an anomaly probably due to deficient development of one renal artery.

The pelvis of the kidney, and the ureter, sometimes present curious anomalies. Sir H. Thompson encountered a kidney with two pelves, which united into a single ureter about an inch below their necks ("Path. Soc. Trans.," vi. 267). In a case recorded by Mr. Wood (Ibid., vii. 261), the left kidney had two ureters, which continued distinct until within an inch of the bladder. The right kidney of the same patient had, in addition to a ureter which entered the bladder at the usual place, an aberrant ureter, connected with a dilatation (partial hydronephrosis) at the upper extremity of the kidney; this aberrant duct was as thick as a goose-quill, sacculated, and opened into the bladder close to the exit of the urethra. Dr. C. Kelly records a case (Ibid., xix. 274) in which no trace of the left kidney could be found; the right kidney had two ureters—the upper one descended in the usual manner; the lower one passed behind it, and, following the course of the left common iliac artery, entered the bladder as the left ureter usually does. There were three arteries, the upper of which was in the usual position of the right renal artery, and three veins; no corresponding vessels existed on the left side.

Horseshoe Kidney.—The most common deviation from the normal shape of the kidney, consists in the fusion of the two organs into one, by an intermediate transverse portion, or isthmus, which connects their lower ends across the spine, so as to form a crescent or horseshoe. Fig. 81 represents a specimen which I removed, some years ago, from the body of a patient who died of phthisis in the Royal Infirmary. The two halves of a horseshoe kidney are usually complete and perfect in themselves, and possess each a separate pelvis and ureter. The transverse portion is generally composed of proper secreting structure; but sometimes it consists merely of condensed fibrous tissue. The concavity of the crescent is nearly always directed upwards; and the ureters generally descend in front of the transverse portion—but sometimes, according to Wilks, behind it. In a drawing possessed by my colleague, Dr. Renaud, the two ureters of a horseshoe kidney are seen to cross each other on their way to the bladder. The arterial supply of a horseshoe kidney always presents some departure from the ordinary distribution.

This deformity does not occasion any derangement in the secretion of urine, provided the organ remain healthy. Rayer reminds practical men, that in thin persons, with flaccid bellies, the transverse portion of a horseshoe kidney may give the feel of a morbid growth in the abdomen; and that suppuration and dilatation of the pelvis of such a kidney may occasion a tumor, which, from its central position near the spine, would lead an observer away from the idea of a pyonephrosis, unless the possibility of this deformity were borne in mind. Neufoille, quoted by Ebstein, describes a case in which congestion of a horseshoe kidney caused pressure on the inferior vena cava, with consequent thrombosis, and death.

FIG. 81.



Horseshoe kidney.

III.—ANOMALIES OF NUMBER.

Rayer cites a number of instances in which there existed one or two supernumerary kidneys, each with its separate excretory duct. The same author cites examples of stillborn infants, presenting a complete absence of both kidneys, together with the ureters and bladder. In acephalous monsters this abnormity appears to be not uncommon.

SOLITARY KIDNEY.—The absence of one kidney has been repeatedly observed in the bodies of persons who presented no derangement of the urinary function during life. The existing organ, in such cases, is always hypertrophied; and so long as it remains healthy, the secretion of urine is carried on without appreciable defect. But if the solitary kidney become inflamed, or its excretory duct obstructed by the impaction of a calculus, or the pressure of a tumor, alarming symptoms make their appearance, accompanied with partial or total suppression of urine, ending in fatal uræmia. *See* Suppression of Urine.

Of twenty-nine cases of solitary kidney collected by me from various sources, 22 occurred in males, 6 in females, and in 1 case the sex is not stated. One was a male infant seven days old; another a boy of seven years; two of the cases were fifteen years old; four were between twenty and thirty, three between thirty and forty, four between forty and fifty, two were sixty, and one was sixty-five—the remainder were adults whose age is not specified. The left kidney was absent in 16 cases, and the right in 12—in one case the side affected is not mentioned. In nineteen of the cases, the defect was congenital; in three, it had been acquired later in life through destruction of the opposite organ; while in seven, it is left uncertain whether the defect was congenital or acquired. The renal vessels and the ureter of the defective side were always absent in the congenital cases. In Dr. Hillier's case, the existing (right) kidney had three arteries and two ureters. The corresponding suprarenal capsule was likewise generally wanting when the defect was congenital.[1] In 24 cases the cause of death was specified: in 12 of these, death was essentially conditioned by the absence of the opposite kidney—and was caused in 10 cases by the impaction of a calculus in the ureter, and in 2 cases by the pressure of a cancerous growth on that canal. In the remainder, the cause of death was unconnected with the anomaly in the kidneys. In a case observed by my colleague, Dr. Leech, solitary kidney was associated with double uterus and vagina.

In addition to the cases collected by Rayer and Mosler, the following examples of solitary kidney may be referred to: Garrod, "Lancet," 1863, ii. p. 724; Rootes, Ibid., 1866, ii. p. 251; Ogle, "Path. Soc. Trans.," 1851–2, p. 382; Sydney Jones, Ibid., vol. viii. p. 279; Murchison, Ibid., vol. x. p. 190; Hillier, Ibid., vol. xv. p. 46; Bruce, Ibid., vol. xvii. p. 175; Kelly, Ibid., vol. xix. p. 275; Duckworth, Ibid., vol. xx. p. 232; Murray, "Brit. Med. Journ.," 1866, ii. p. 159; Taylor, Ibid., 1870, ii. 485; Meschede, "Virch. Arch.," Bd. 33, S. 546.

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[1] Beumer, however, found the suprarenal body absent in only 5 out of 48 cases. He also points out that the sexual organs are frequently arrested in development on the same side on which the kidney is wanting.

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